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SYMPOSIUM ON HEADACHES.

HEADACHES DUE TO THE EYE.*

DR. W. G. GILLETT, Wichita, Kan.

The symptom complex which we now designate as asthenopia, with its associated headache, was accurately described by Taylor in 1776 and by Plenck in 1792. The weak eyes which baffled all their efforts for relief was described by them as brow pain. Prior to 1850 the condition had been written on many times by such careful observers as Scarpa, Beer, Lawrence and others. Good, in his work entitled, "The Study of Medicine," published in 1829, has the following to say regarding chronic headaches. "It is frequently experienced by hard students, who have sat up through the whole of the night in pursuit of some abstruse and difficult subject, or who have labored upon the same from week to week with too small an allowance of time for sleep or exercise. In all which cases it is often relieved by surrounding the temples with a bandage steeped in cold water, which acts as a tonic upon the spent and enfeebled brain and once more excites it to a little temporary energy." The variety of opinions by these eminent men as to the true nature and cause of the condition and the different treatments proposed, convict them of their error. It was not until 1858 when Donders published his classical treatise on the "Anomalies of Refraction and Accommodation," that the obscure brow pain of the older observers began to fade out, and even then the true nature of headaches due to eye strain were not clearly estab-

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lished until the publication of William Thompson in 1871 and S. Weir Mitchel in 1874.

It has been estimated by various authorities that 40 per cent of all headaches have an ocular factor, and that 75 per cent of all bilateral frontal headaches are due to eye strain. These figures may seem high but it is certain that headache is probably the most common symptom encountered in the practice of ophthalmology. Zimmerman found that in a series of two thousand cases 71 3/10 per cent complained of headache. The subject should therefore assume a commanding importance in our field of medicine.

There are innumerable classifications for headaches, but those of the internist, neurologist and other specialists do not fit in well with the working scheme of the oculist. Lancaster has classified headaches from an ophthalmological viewpoint under two headings. First, those in which an ocular condition is the cause of the headaches, such as eye strain, glaucoma, various forms of conjunctivitis, iridocyclitis, scleritis, local inflammatory conditions of the lid, etc. Second, those in which the headache is not ocular in origin, but something about the eye betrays the true nature of the cause of the headache. Thus, optic neuritis, retinitis, retinal hemorrhages, arterosclerosis of the retinal vessels, papilledema, diplopia, pupillary changes, defects of the visual fields, etc., often throw light on the condition of which headache is only a symptom.

Of the first group, eye strain is by far the most important and it is this subject which will receive the most attention in this short paper.

The term "eye strain" is a useful one, but there are so many different explanations for this condition that few of us have the same ideas regarding it. Reilly would have us believe that in the last analysis all the pain of eye strain is due to spasm of the ciliary muscle. Others feel that fatigue and hyperesthesia of the retina are among the salient factors, while still others believe that muscular imbalance is more important than anything else. According to Lancaster it is the adjusting mechanism of the eye that is strained and this adjusting mechanism is composed of two parts; first, the focusing mechanism, which secures a clear image on the retina; and second, the fixation mechanism, which fixes the eye on the object to be looked at. He also points out that eye strain is more than fatigue or strain of this mechanism, for it has been shown that the extrensic muscles are capable of pulling one hundred times the load ever required in ordinary eye movements and we know that there are thousands of cases of hyperopia, of quite large degrees, who are absolutely comfortable without correcting glasses. This other factor which enters into eye

strain is the fatigue of the elaborate and intricate system for co-ordinating these muscles, *i. e.*, fatigue of their motor nerve cells and centers.

Now, if we agree that fatigue or strain of one or all of the above mechanisms can produce headaches, then under what conditions do these headaches become manifest? They most commonly become manifest when the normal mechanism has some handicap, such as a refractive error, to contend with. The refraction in one eye may be different from the other. The patient may be recovering from some sickness, operation or general debilitating condition in which all the muscles of the body are weakened. Undue strain may be placed on the mechanism because of poor light, small print or an unsteady object to be focused. The media of the eye may be clouded or the retina diseased, thus throwing a more constant effort on the focusing mechanism in an effort to secure a clearer picture. These are only a few of the many exciting causes which might be mentioned.

Is there any way to say positively that a given headache is ocular in origin? I do not think so. It is true that most ocular headaches are frontal and bilateral, that they follow long continued use of the eyes in a poorly lighted room, etc., but eye headaches have been described as low down as the twelfth dorsal vertebra and in every conceivable place on the head. Many other conditions have produced headaches in exactly the same places. Some authors would be very specific about eye headaches and give them a definite order of frequency, such as *1.* supraorbital, *2.* deep orbital, *3.* fronto-occipital, *4.* temporal. Kearney would even go so far as to classify the refractive error by the position of the headache, a frontal or supraorbital headache indicates a hyperopic error, an occipital headache an imbalance of the extrensic muscles, and temporal headache an astigmatic error. A headache occurring in the frontal and temporal region would, therefore, suggest a compound hyperopic astigmatism, etc.

Ocular headaches are not often encountered early in the morning, though there is one form which is supposed to be due to sudden spasm of the ciliary muscle on first opening the eyes. This is seen in people who have used their eyes far in excess of normal on the preceding day. Myopic patients, of course, have very few headaches which are directly attributed to their eyes.

Of the other headaches which were purely ocular in origin, glaucoma is the next in importance and I shall not have time to touch on those due to the inflammatory conditions.

The headaches of glaucoma are important because they are often in the occipital region and so insidious that the patient does not asso-

ciate it in any way with his eyes. The condition is rare enough so that it is not thought of by the general practitioner and is very often overlooked by the experienced ophthalmologist. These cases are usually past middle life, the course is chronic and is very frequently associated with nephritis or a mild arteriosclerosis, so that the attending physician is perfectly willing to attach the headache to one or the other of these more frequent causes. When these stubborn cases are encountered they should of course be sent at once to the oculist, where the ophthalmoscopic findings, fields and tension can be obtained and the proper treatment instituted.

In the second group there are a number of interesting conditions where the headache is not ocular but something about the eye betrays the true cause of the headache; only a few of these conditions will be taken up.

Bitemporal headaches, not unlike the headaches of eye strain, are among the earliest symptoms of tumor of the pituitary gland and this symptom alone may bring the patient to the oculist. On fundus examination he may find a pale optic nerve without loss of substance and characteristic field changes which help make the diagnosis.

In a similar manner patients with headaches due to a brain tumor often find their way to the ophthalmologist, where the diagnosis is often confirmed by papilledema, field changes, muscle palsies, etc.

Without wishing to trespass into the field of the laryngologist I would like to call attention to the so-called vacuum frontal headaches with eye symptoms. These cases have no nasal discharge or other nasal symptoms to send them to the laryngologist and their headaches are always aggravated by reading or doing close work, consequently they feel that their eyes are the offending organs and are often fitted with glasses which give no relief.

Infections in the frontal and anterior ethmoidal cells occasionally give similar symptoms but infections in the sphenoidal cells are seldom brought to the attention of the oculist unless it has produced an optic neuritis.

In closing it might be well to emphasize the fact that in headaches arising from affections of the nose the symptoms are often described as being worse the first thing in the morning and improving later on in the day. Just the opposite is true of most headaches developing from eye strain; they are comparatively free from headaches in the morning but as the day wears on their headaches develop and are worse in the late afternoon.

201 N. Main Street.

THE NASAL ASPECT OF HEADACHES.*

DR. JAMES C. BRASWELL, Tulsa, Okla.

In the consideration of nasal headaches one is forcibly impressed with the illuminating work of Sluder. His researches in connection with the anatomy and pathology of the nose, its accessory sinuses and the nasal ganglion, form the most important contribution to the subject in rhinologic literature.

The symptoms of headaches may in some instances be so marked as to obscure the less evident symptoms and to prompt both the patient and physician to mistake the headache for the disease itself. Of the more common forms of headaches, practically all are correctable if the cause is ascertained and then removed.

Pain may be regarded as a reaction of the organism in part or as a whole, to harmful influences giving a warning in consciousness that some activity prejudicial to the health of the tissue is operative. Biologists claim that the movements of expansion and contraction of protoplasm are primordial expressions of pleasure and pain sense, expanding to pleasure, giving and contracting to pain giving or harmful impulses, hence any agency in the system, which through pressure, chemical or irritation causes undue contraction of the nerve protoplasm, tends to cause pain. Pain sensations are transmitted to the brain centers through the afferent or sensory nerves and the pain is referred to the origin of the nerve fiber, whether the irritant is applied to the periphery or to any part of the nerve in its course to the brain.

The sensory nerve supply of the nose is derived from the first and second divisions of the trigeminus. The anterior ethmoid nerve, a branch of the first division, supplies the upper anterior part of the nasal chamber and sends fibers to some of the anterior ethmoid sinuses and frontal sinuses. The remainder of the surface of the nose and accessory sinuses derives its sensory innervation through the sphenopalatine ganglion from the second division. Motor and sympathetic fibers also pass through this ganglion from the Vidian nerve, which is made up of fibers from the great superficial and deep petrosal nerves. The sensory fibers are distributed not only throughout the greater part of the nasal cavity, including filaments to the upper anterior part of the nasal chamber supplied by the anterior ethmoid nerve, also to the orbit, palate, nasopharynx, tonsil and tongue. Motor fibers of the lacrimal nerve also pass through this ganglion.

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Bearing in mind the nerve supply of the nose, it is easy for one to reach the conclusion that lesions in the upper anterior part of the nasal chamber and the anterior ethmoid and frontal sinuses affect the anterior ethmoid terminations and produce pain referred to the distribution of the first division of the fifth nerve. On the other hand, lesions in other parts of the nasal cavity which are in close relation to the sphenopalatine ganglion and its afferent and efferent trunks, may produce pain referable to the distribution of the second division.

Greene is of the opinion that conditions in and about the nose which may give rise to headaches and neuralgias may be grouped into four classes:

1. Those in which pain is due to acute inflammation in the nose or paranasal sinuses.

2. Those in which chronic inflammation in these regions is the etiologic factor.

3. Anatomic variations in structure which result in pressure contact within the nasal cavities.

4. New growths of the nose and accessory sinuses.

Pain in acute sinusitis is best explained on the basis of pressure upon the sensory nerves. The pressure may be due to swollen tissue or a collection of pus in the sinus or combination of both.

The pain of an acute sinusitis is characterized by the daily occurrence. In very severe cases with suppuration in which there is no drainage, the pain may be constant; however, it is usually more intense during the forenoon, abating during the afternoon and evening. The location of the pain depends upon the sinus involved. In acute frontal sinusitis the pain is located in and about the eye and sinus, combined with tenderness above and behind the inner canthus. The pain in acute maxillary sinusitis is located in the cheek and upper jaw and may be referred to the teeth, eye, supraorbital region and, in instances, to the ear. In acute ethmoiditis and sphenoiditis the pain is usually quite intense and apparently deep-seated. The area involved is that supplied by the nasal ganglion. In some cases the pain may be referred to some remote region, such as the occiput, ear and shoulder. Sluder has repeatedly called attention to the fact that inflammation in the posterior ethmoid and sphenoid region affects the nasal ganglion or its branches and produce pain in the region supplied by sensory fibers passing through the ganglion and remote regions reached through the sympathetic fibers which pass through the Vidian, by the great deep petrosal nerve, to the carotid plexus, thence to the cervical sympathetic. In order to accept Sluder's theory it is necessary to establish the fact that the sympathetic system has a sensory function. If this theory is accepted and proven it would offer

satisfactory explanation for relief from pain in remote regions following cocainization of the nasal ganglion.

In cases of chronic frontal and maxillary sinusitis pain is relatively infrequent except during acute exacerbations, when it simulates the acute conditions.

The diagnosis of acute inflammations of the sinuses is relatively simple and can be made from the history, intranasal inspection, transillumination and X-ray. In chronic inflammations of the anterior ethmoids, frontals and maxillary sinuses, the diagnosis is usually simple. In the posterior ethmoids and sphenoids the problem is more complex and it is frequently necessary to apply all means of examinations at our disposal. Cocainization of the nasal ganglion does not relieve pain from a lesion in the sphenoid sinus, but it may be relieved by cocainization of the sphenoid sinus. Due to the wide variation in the interpretation of pain in this area, one is justified in proceeding with caution in an effort to determine the character, type and location of pain presented for diagnosis. Due to the illuminating work of Sluder one is prone to err in the diagnosis of a simple local lesion as the direct cause of pain, whereas further study will often furnish evidence of sufficient weight to class the pain as purely functional. The so-caled psychoneurotic type present pains of various types and only diligent study, combined with all of the mediums at hand, will definitely determine the correct diagnosis. Co-operation of the internist and neurologist is essential in cases of this type.

Headaches resulting from anatomic variations in the nose causing pressure contacts are by no means uncommon. Pressure headaches resulting from deviations or thickening of the posterior part of the septum or enlarged turbinate occur with frequency. The cause of such contacts may be traceable to acute inflammations or external irritants. The vacuum headache described by Sluder necessarily falls under this type. In many instances marked pressure contact is present in the nose without any evidence of pain and the logical deduction is that the local condition is only a factor in the cause of pain.

New growths in the nasal cavities may be the cause of pain; however, malignant conditions of the sinuses frequently reach the inoperable stage without the slightest manifestation of pain. It is by no means uncommon to find the nasal cavities filled with polypoid growths of such size as to materially interfere with breathing and phonation, and absence of pain prove to be an outstanding feature.

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Mayo Building.

HEADACHES OF INTRACRANIAL ORIGIN.*

DR. FRANK R. TEACHENOR, Kansas City, Mo.

Permit me to express my appreciation of the honor of appearing as a guest of the Kansas City Eye, Ear, Nose and Throat Society to participate in this symposium on headache.

As the subjects of toxic, reflex and other headaches from ophthalmic, nasal, otitic, infectious, endocrine and general constitutional diseases are to be covered by the other essayists of this symposium, you will permit me to enter immediately upon a discussion of headache from actual intracranial disease.

Headache or cephalalgia is almost a universal symptom; appearing, as it does, from such a multitude of diversified diseases throughout the body. It is peculiarly common to intracranial conditions and appears almost invariably in some stage of these diseases. Headache alone is rarely diagnostic of intracranial disease. It is its association with neurological and other physical symptoms that a diagnosis, and particularly a localization of intracranial disease is possible. However, with a knowledge of the nerve supply of the cranial contents, headache may be very helpful in diagnosis in the absence of definite localizing signs.

While the brain is the perceptive center of all sensation, it is apparently insensitive and incapable of initiating painful stimuli. It may be cut at operation without pain to a conscious patient. The meninges are, however, quite sensitive to pain, particularly to stretching and compression. It is from the meninges, particularly the dura, that headache is derived.

The nerve supply of the pia is chiefly from the sympathetic, and accompanies the arteries. It also receives some afferent fibres from the third, fifth, sixth, seventh, ninth, tenth and eleventh cranial nerves.

The arachnoid has no demonstrable nerve supply.

The dura has a rich and somewhat complicated nerve supply. It is supplied mainly from the fifth and tenth cranial nerves and the sympathetic, although some fibres are derived from the fourth and twelfth cranial nerves.

The ophthalmic division of the fifth, through its anterior and posterior ethmoid nerves, supplies the dura in the frontal region; and a

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recurrent tentorial branch, with some filaments from the fourth cranial nerve, supplies the tentorium cerebelli. This accounts for the reflex frontal and supraorbital headache encountered in lesions of the posterior fossa compressing and elevating the tentorium.

The superior maxillary division of the fifth nerve, through its meningeal branch, and filaments from the Gasserian ganglion supply the dura of the base of the middle fossa.

The mandibular division of the fifth nerve supplies the remainder of the dura of the middle fossa and parietal region through its meningeal branch, the nervus spinosis. This branch is given off directly after the exit of this division from the foramen ovale and passes backward to re-enter the cranial cavity through the foramen spinosum, along with the middle meningeal artery. It divides with the middle meningeal artery into an anterior and posterior branch and accompanies these arteries to supply the dura. The posterior branch also supplies mucosa of the mastoid cells.

The dura of the posterior fossa is supplied by a recurrent branch from the sensory ganglion of the tenth cranial nerve (Vagus), with a small recurrent branch from the twelfth cranial nerve. The nerve supply of the posterior fossa, being mainly from the Vagus, accounts for the reflex occipital headaches encountered with abdominal and pelvic conditions. Likewise the close association of the Vagus with the sympathetic, spinal accessory and hypoglossal nerves accounts for the dysphagia, discomfort and rigidity of the neck encountered with lesions of the base and posterior fossa.

Sensation to the scalp, anterior to the vertex, and the face is supplied by the fifth cranial nerve; the scalp posterior to the vertex being supplied by the first four cervical spinal roots.

Headache of intracranial origin is chiefly the result of meningeal irritation or compression. The first will be referred to as irritative and the second as mechanical. They may appear separately, but are frequently present in combination.

Irritative headache is the result of early localized inflammatory reaction, vascular disease, brain tumors, etc., which cause dural irritation. The dura in contact with adjacent infected paranasal sinuses, infected mastoid or osteomyelitis of the skull through irritation or early inflammatory reaction causes headache. Varicosities, aneurism or sclerosis of the pial or dural vessels have similar irritative effects; as do also tumors of meningeal or cortical origin. Exostosis of the inner table of the skull or other osteal tumors, cortical or meningeal scars or any other condition which causes meningeal irritation, without evidence of increase of intracranial pressure, may be included.

The character of this type is a more or less localized headache, of a dull, sharp or boring type. More commonly it is a diffuse headache with increased intensity at the site of the local irritation. Frequently the scalp is painful or sensitive to pressure over the site of local irritation, and percussion over this area increases the intensity of the headache. It may be continuous, periodic or intermittent.

As these etiological factors increase in extent, the headache tends to become more diffuse and of greater intensity. This is particularly true of tumors and inflammatory conditions with localized meningitis, extradural, intradural and brain abscesses. Soon an increase of intracranial pressure exists and the mechanical factor enters.

Mechanical headache is the result of a change of intracranial pressure. Any change in intracranial pressure may cause headache.

A reduction of intracranial pressure may produce headache, as is illustrated by the "so-called" spinal puncture headache which follows the rapid withdrawal of an excessive amount of cerebrospinal fluid. This headache is credited to loss of the water bed in the basilar cerebrospinal fluid cisterns, so that the weight of the brain rests upon and compresses the nerves of the dura of the base, where the nerve supply is very rich. It is a diffuse, throbbing headache, with a tendency to increased intensity in the frontal and occipital regions. A reclining position with lowering of the head gives relief.

Headache from increase of intracranial pressure is well known. It is a very common occurrence with brain tumors and cysts, tumors of the inner-tables of the skull meningeal and vascular tumors, brain abscess, cerebral edema, intracranial hemorrhage, brain trauma, meningitis, encephalitis, sinus thrombosis, hydrocephalus or any other condition which may increase intracranial pressure. The headache characteristic of this condition is a deep, diffuse pain which increases in severity as the intracranial pressure increases and has periods in which the pain is throbbing or pulsating. It also has a tendency to increased intensity in the frontal and occipital regions. When the intracranial pressure is high the throbbing increases and the patients frequently state that the head feels as though it would burst. Sneezing, coughing, straining, etc., increase the intensity of the headache by suddenly increasing intracranial pressure. This headache may be continuous and of increasing severity but peculiarly it tends to become paroxysmal and intermittent. Exceedingly high pressure tends to render the patient stuporous or unconscious, but in instances where the intracranial hypertension is prolonged the headaches may subside before stupor or coma intervenes. This is probably the result of pressure inhibition of the pain fibres or centers.

The intermittent feature of the mechanical headache is interesting, and is common in patients with prolonged increase of intracranial pressure, as with brain tumor. With lesions in the anterior and middle fossae, without hydrocephalus, it is probably due to variations in the blood supply, with resultant variations in the amounts of cerebrospinal fluid or cerebral edema present. In lesions of the cerebellum or posterior fossa the flow of cerebrospinal fluid through the aqueduct of sylvius or the fourth ventricle is interfered with by pressure. This produces a dilitation of the cerebral ventricles, an internal hydrocephalus, with an increase of the headache. When the pressure in the cerebral ventricles becomes high enough to force a passage through the compressed aqueduct and empty the ventricles the intracranial pressure is reduced by absorption of the fluid and the headache subsides. This occurs in cycles and accounts for the intermittent character of the headache.

When these two types of headache exist simultaneously the severe diffuse throbbing headache of compression may be modified by the localizing character of the irritative type. It is very difficult for patients to discriminate between the various types of headache, or to describe their pains in an intelligent manner. Headache being a subjective symptom, we are entirely at the mercy of the patient to render this symptom helpful or useless.

The scope of this paper and the time allotted will not permit a discussion of the physical and neurological signs associated with headache necessary to a localization of brain lesions. I will merely mention some of the variations in headache in lesions of different locations.

Frontal lesions (including tumors, abscess, etc.) produce diffuse headache, with increased intensity in the frontal region and frequently referred pain to the occiput. In frontal lobe abscess the headache is an early sign, while in frontal tumors it may not appear until late. This is particularly true of glioma, which absorbs and replaces brain tissue, thus causing an increase of intracranial pressure only after the tumor has reached a considerable size. This is equally true of gliomata elsewhere. Tumors which displace and compress brain tissue produce headache and intracranial pressure earlier. Choked disc is a late sign in frontal lesions. Motor aphasia and signs of involvement of the motor areas may occur.

Lesions of the cerebellum and posterior fossa usually produce headache as an early sign. The choked disc may appear early and show the highest degrees of swelling. The headache is occipital and projected through the center of the head to the frontal region. Dif-

fuse throbbing headache accompanies it. There is usually some distress, or stiffness of the neck. The presence of cerebellar atoxia, vertigo and nystagmus complete the picture.

Lesions of the middle fossa produce headache of the diffuse variety, with increased intensity in the temporal regions. There may be headache of greater intesity on the side of the lesion or bitemporal headache. Deep orbital headache occasionally occurs. The neurological signs are diversified, hemiplegia and aphasia being frequent. "Uncinate fits" and the "dream state" occasionally accompany lesions deep in the temporal lobes.

Pituitary headache is of a deep, bursting, bitemporal character, and frequently there is much pain deep in the orbits. Hemianopsia, enlargement of the sella tursica, and pituitary dyscrasias may be present to some degree.

Lesions of the parietal and occipital regions produce diffuse pains, occasionally of greater intensity near the vertex. Frontal headache is not infrequent. Lesions of the parietal lobes are characterized by paresthesias, ostereognosis and at times involvement of the motor area. The homonomous hemianopsias characterize tumors of the occipital lobes.

Tic douloureux is characterized by sharp, paroxysmal, lightning-like pains, which shoot through the areas supplied by one or more divisions of the fifth cranial nerve. While headache may accompany it, the pain is more common in the peripheral distribution in the nerves in the face, temple and forehead. When it is caused by compression of the Gasserian ganglion, as in brain tumor, the neuralgic pains are accompanied by headache deep in the temporal region and orbit, frequently unilateral, occasionally diffuse. Infections of the paranasal sinuses and teeth cause pains which closely resemble tic douloureux, and must be ruled out before a diagnosis of tic douloureux is made, especially before the radical operation for its relief is done.

The headache of meningitis is very intense, diffuse and throbbing in character and referred to the frontal region. Later there is occipital headache, with rigidity of the neck muscles, Kernig's sign, opisthotonus and purulent cerebrospinal fluid. The headache of syphilitic meningitis is particularly active at night.

Increased intracranial pressure causes headache, nausea and vomiting. When pressure continues over a few days papilledema and choked disc appear, which may go on to optic atrophy. These signs were termed the cardinal signs of brain tumor, but are only general signs of intracranial pressure.

The headache of cerebral edema is typical of the diffuse mechanical headache. Cerebral edema is a common condition. It accompanies many of the intracranial lesions previously discussed. It commonly occurs with nephritis, cardiovascular disease, eclampsia, alcoholism and numerous constitutional diseases. Many of the toxic headaches occur as a result of cerebral edema.

The differential diagnosis as to the character of the lesion, as you all know, may exhaust all of our physical, radiological, serological and laboratory resources to determine. It must be remembered that headache is only a symptom. It is rarely ever diagnostic in itself, merely suggestive.

Argyle Building.

TREATMENT OF OBSTRUCTING INFERIOR TURBINATES.

DR. M. L. HARRIS, Brooklyn.

Nasal obstruction is a very common complaint for which patients seek relief. It may be due to a septal deviation but more frequently to an obstructing inferior turbinate. The textbooks describe various methods of handling hypertrophic inferior turbinates, which are sometimes part of a chronic hypertrophic rhinitis, sometimes compensating with a deviation of the septum and often as a result of venous stasis. The treatment should take under consideration the difference in pathology.

Inferior turbinates may hypertrophy from diseases above. Discharges from the ethmoids and other sinuses will secondarily affect the turbinates, causing vasoparesis in the erectile tissue. At the beginning the obstruction will be intermittent, depending on variations in the rest of the nasal mucosa and the character of the discharge. This gives us the so-called turgescent inferior turbinate. Finally, the obstruction may be constant when hyperplasia takes place in cases of chronic inflammation. The inferior turbinate that shrinks little under

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adrenalin is probably hyperplastic, and the one that shrinks to half or less is turgescent and gives intermittent obstruction.

It is manifestly unsound to remove turbinates completely except for malignancy. What we need do is to restore function. All that is necessary is to give the patient sufficient breathing space to make him comfortable and provide proper aeration for all the nasal chambers.

The obstruction may be handled in various ways, depending on its cause and its pathology. An inferior turbinate which is hyperplastic is best partially removed and a simple method is that with the clamp and cautery, as this is not followed by troublesome bleeding.

Submucous resection involves removal of the turbinate bone and is rather a long procedure and not always easy. Cauterization only destroys the mucosa.

The inferior turbinate that causes intermittent obstruction is one in which the venous channels get engorged intermittently and these are best treated by coagulation of these venous spaces. This is best accomplished by the high frequency current. The monopolar method is used and coagulation is produced by inserting the needle for distances varying from ¼-inch to ½-inch at various points in the submucosa and the current allowed to flow for about ten seconds. This is repeated in a week and again if necessary until the turbinate assumes a stationary position without obstruction. Of course, disease of the sinuses which is frequently the cause of this condition should also be treated at the same time.

This method of reduction of an hypertrophied inferior turbinate is simple, there is no bleeding, requires no packing and there is no danger of postoperative synechia, as in repeated cauterization.

1681 Union Street.

A PLEA FOR CONSERVATISM IN THE TREATMENT OF OPTIC NERVE DISTURBANCE FROM FOCAL INFECTION.*

DR. LEON E. WHITE, Boston.

Needless or too radical intranasal surgery in cases of optic nerve involvement from focal infection is the subject presented for your consideration. The data is derived from my last 60 unpublished cases. Some of these were seen only once in consultation, but as the results are known they have been included in my series. In my absence several were followed by my son. The few that failed to carry out my recommendations are incomplete.

I hope to show from this study that it is usually possible to find the focus of infection and by eliminating it before the nerve is damaged, to obtain prompt recovery. It will be shown that in the majority of the cases only the focus needs to be eliminated, although it is advisable to remove nasal obstruction when well marked. In but few cases has it been necessary to open the posterior sinuses, and even in some of these it was needless or done too late. The routine opening of uninfected sinuses has been found unnecessary and is to be condemned.

The papers constantly appearing on this subject I regret to say nearly all advocate too radical intranasal surgery. While some writers merely recommend a unilateral ethmoid and sphenoid exenteration, others are only satisfied with a bilateral one, to which in some instances is added a tonsillectomy. I have been expecting that in addition to the bilateral exenteration of the sinuses and the removal of the tonsils, someone would advocate extraction of all the teeth. This would seem about as sane as the wholesale exenteration of uninfected sinuses. Teeth that often excel in beauty and utility might replace those extracted but as yet nothing has been devised to replace the sinuses.

Before becoming too critical, let me admit that in my early studies sinuses adjacent to the optic nerve seemed the all-important foci of infection and that by draining them the cases would be cured. My first 17 cases were attributed to the sinuses. Most of the papers appearing at the present time still maintain this view. This is largely due to the fact that the opening of the accessory sinuses is done about

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the time the case would normally commence to improve or to the benefit following the depletion of the structures adjacent to the nerve. Teeth or tonsils are rarely mentioned.

Volumes were written on the anatomic relationships of the optic nerve to the posterior sinuses, and while I should hesitate to say that none of this study was valuable, for much of it was enlightening, still I do maintain that it threw sinuses too much into the limelight, while the more remote sources of infection were decidedly left in the background.

In my effort to find sufficient pathology in the sinuses to account for the optic nerve disturbances, I had careful examinations made of the tissues removed from the sinuses and turbinates. While this tissue frequently showed changes, it failed to convince the pathologists that it was sufficient to account for the eye conditions. More remote sources of infections were then sought and soon tonsils presented a focus which loomed up as an important etiologic factor.

From tonsils it was but a step to teeth, so I began to have the teeth filmed in all cases. Along with the teeth and tonsils the antrum took on added significance, not as a sinus from which there might be direct extension to the optic nerve, but as a focus from which infection might pass by way of the blood stream or lymphatics to the nerve. At first it was the antrum with pus that was especially sought, but later marked thickening of the mucosa seemed fully as important.

At present as I look in retrospect over more than 100 cases, it seems that the infection comes either from some systemic condition, as influenza or intestinal toxemia, or from some definite focus, such as teeth, tonsils or sinuses; that the infection travels by way of the blood stream and produces a neuritis of the optic nerve the same as of any other nerve. Eliminate the infection from the blood stream and the neuritis subsides. The danger lies in the fact that as the optic nerve is a highly specialized structure it degenerates easily and never regenerates. There is always a possibility that in a severe neuritis optic atrophy may intervene before the inflammation subsides. To this end let us consider the type of case where this might happen and the methods of combatting it. It is a well recognized fact that the majority of the cases of acute optic neuritis recover spontaneously so that if there were some method of distinguishing betwen those that might reasonably be expected to recover from those that might not, they could at once be dismissed.

As to the methods of differentiating the cases, the duration and extent of the loss of vision, the condition of the fundus, the virulence of the infection and the size of the optic canals must all be considered.

Duration: The conclusions reached from an analysis of the cases were that unless there was improvement under treatment within a week there was danger of permanent impairment of vision, while in cases of more than two months' standing little could be expected except possibly checking the loss of vision by the removal of some definite focus of infection.

The Amount of Vision: It was also found that the demand for early operative interference in total loss of vision was more imperative than when the loss was but partial.

Condition of the Fundus: When the nerve appears normal one might, with safety, delay operating much longer than where there is increasing engorgement or commencing pallor.

The Virulence of the Infection: As in all the other types of infection, so in that producing optic neuritis, the micro-organisms differ greatly in virulence. When the infection is of the virulent type there is probably considerable exudate about the nerve or even within its sheath. The optic nerve, as you know, is really not a nerve but a part of the brain. It is easily destroyed and does not regenerate. Parsons says of it: "The so-called optic nerve, together with certain parts of the retina, constitutes a lobe of the brain, and has, therefore, the characteristics of the central nervous system. Hence the nerve fibers are devoid of a sheath of Schwann and the interstitial substance is neurolgia."

The Size of the Optic Canal: That the osseous optic canals normally afford great protection to the neural contents cannot be doubted but when these canals become narrowed and the enveloping sheath of the nerve, or the nerve itself, swollen, the unyielding walls may cause constriction sufficient to produce atrophy.

Many ophthalmologists have maintained that most cases would have recovered if nothing had been done and have intimated that the brilliant results following intranasal surgery would have taken place had nature been permitted to work unhampered by the enthusiastic rhinologist. The fact remains, however, that blindness does follow in a certain number of these cases. The problem is to separate the cases where spontaneous recovery might be expected from those where permanent damage would follow if untreated. Rhinologists have been criticized for operating on all cases irrespective of their severity. They justified these procedures by claiming that by these methods they would relieve those cases where blindness might follow if untreated and maintained that these operations were without danger. It soon became apparent, however, that bilateral exenteration of the ethmoids, for instance, was not devoid of danger.

Cushing, having seen several cases of meningitis following ethmoid exenterations, did us all a valuable service in calling attention to these fatal results and halted somewhat the tendency for too radical and too irrational intranasal surgery. The word irrational refers to the numerous cases he reports where intranasal surgery was performed for the relief of blindness due to brain tumors, a sad commentary on the lack of diagnostic skill on the part of the rhinologists.

To return, however, to the original discussion—Is it possible after a careful study of these cases to differentiate between them? To a considerable extent, yes. To an absolute certainty, no. There are cases so slight and transitory that it would be little short of criminal to submit them to any surgical procedure not indicated in our ordinary rhinological findings. Infected teeth or tonsils should be removed. If the antrum shows infection it should receive appropriate attention. But to exenterate ethmoids and sphenoids not so evidently diseased that they require opening irrespective of the eye condition, is, I believe, a gross but too common error. Mild cases recover without treatment or with ordinary astringent sprays. These cases are usually taken too seriously. Extreme measures are instituted where little or no treatment would suffice, and while recovery may take place in spite of, rather than because of, these procedures, the patient is submitted to needless risks and the intranasal structures are unnecessarily mutilated. Then again there are cases with marked loss of vision, yet with a normal optic canal. Here again the focus should be diligently sought and, where possible, removed. Should the posterior sinuses be markedly obstructed by an enlarged middle turbinate or a deflected septum, aeration of this region is demanded by the removal of the obstruction. In the ordinary run of cases reliance should be placed on hot alkalin douches, astringent sprays, suction, etc. These first two classes probably comprise 85 per cent of all the cases arising from focal infection and recovery in all should be obtainbed by conservative methods. In the remaining 15 per cent where we are contending with a severe neuritis with marked or complete loss of vision, especially in a patient with an optic canal considerably below normal, I believe that in addition to the removal of the focus it is usually advisable to also remove the middle turbinate, take down the front wall of the sphenoid and uncap the posterior ethmoid cell. This procedure is especially advocated if the blindness is of several days' duration and the fundus shows increasing engorgement or commencing atrophy. In none of the cases do I advocate exenteration of any of the other sinuses unless they show pathology sufficient to warrant it, irrespective of the eye condition. Since I have followed the procedures above indicated my

results have been as good as when, as a matter of routine, I opened the posterior sinuses.

So much has been said and written on the intimate relationships between the optic nerve and the sinuses that it has become almost an obscession with rhinologists to consider the sinuses, even if normal, in some mysterious way responsible for the eye disturbances. An explanation of this mystery was attempted in a paper I presented before the American Laryngological Association in Montreal in June, 1926, entitled, "The Influence of Negative Pressure in the Sphenoid On the Optic Nerve." In this article it was explained that the benefits usually following the opening of the posterior sinuses resulted from the relief of the negative pressure and not from the removal of a focus of infection.

Onodi's writings on the contralateral relationship of the optic nerve to the posterior sinuses has undoubtedly led to the needless bilateral exenteration of many normal sinuses.

At the Dallas meeting of the American Medical Association in 1926 several extremely interesting papers on iritis were read before the Ophthalmological Section. A paper by Iron and Brown, entitled "Recurrence of Iritis As Influenced By the Removal of Infections," deals with fifty cases of iritis followed from three to twelve years. The probable source of the infection was found in forty-six of these cases. The teeth alone were considered the focus in 20 per cent; the tonsils in 34 per cent; the sinuses (antrum) in 2 per cent; the teeth and tonsils combined in 10 per cent; the sinuses, teeth and tonsils in 4 per cent; and the sinuses, teeth and colitis in 2 per cent. Thus, it would seem that sinuses play as minor a role in iritis as they do in optic neuritis.

Zentmayer in a paper entitled, "The Prostate As a Remote Focus of Infection in Ocular Inflammations," directed attention to the importance of searching further than the mouth, nose and throat for the portal of infection. He states that it is not infrequently found in the prostate gland.

A third paper by Mills, entitled "Ocular Disease Occurring in the Course of Nondysenteric Amebiasis," calls attention to the importance of investigating the intestinal tract. He states that "it is certain that the arrest or cure of chronic iridocyclitis, choroiditis, retinitis, episcleritis, keratisis and conjunctivitis has followed the faithful use of ipecac and its derivatives when the recognition of a protozoan or flagellate background has ended repeated vain searches for a bacterial etiology." He furthermore states that in his sole case of double optic atrophy three varieties of endamebas were found at the first exam-

ination of the stools, "and that all other tests, including Roentgenograms of the brain, sella and sinuses were without results."

It may interest you to know that Dr. George Derby's treatment for retrobulbar neuritis when no definite focus is found is to put the patient in bed, sweat him, flush out the kidneys and intestinal tract. That the appendix and gall bladder may be sources of infection to the blood stream is now well recognized. One of my cases of retrobulbar neuritis was attributed to infection in the pelvis.

It is imperative that one who studies optic nerve lesions should be conversant with all possible sources of infection. Owing to their propinquity the sinuses have long shouldered the responsibility for most of these disturbances. This burden must now be distributed between the teeth, tonsils, sinuses, prostate, appendix, gall bladder, Fallopion tubes and intestinal tract.

You may be interested in our method of investigating cases: Patients are always referred from either an ophthalmologist or from an ophthalmological clinic, so that the eye report usually accompanies the patient. This should give a history of the case, its duration, vision, visual fields when possible, and condition of the fundus. A correct diagnosis is naturally essential before surgical intervention is advised. The symptoms in the ordinary cases of acute optic or retrobulbar neuritis are quite typical. Associated with the loss of vision there may be discomfort about the eye or lameness on moving it. Occasionally there is slight exophthalmos, this condition indicating an inflammatory process in the orbit. Pupillary changes and ptosis are found occasionally. There is frequently nyctalopia, a central scotoma for colors, enlargement of the blind spot and contraction of the fields. The patients generally say that things at first were blurry when looking straight ahead, but comparatively clear when viewed sideways. Changes in the fundus are of great diagnostic value, varying, as they do, from the normal to a commencing pallor in some cases, and in others to a marked redness and swelling about the nerve, with engorgement and tortuosity of the retinal veins. It is essential that these inflammatory swellings be differentiated from the edema, due to intracranial pressure.

A careful examination is made of the nose, throat and mouth, the sinuses transilluminated, and the position of the septum and size of the middle turbinates are especially noted. The anterior portion of the nose frequently appears normal. The one vital point to determine, in the nasal examination, is the size and position of the middle and superior turbinates. Do they block the ventilation of the posterior sinuses? Is there impaired aeration? It takes but a slight obstruction to interfere with the ventilation, and the mistake is fre-

quently made of expecting to discover marked changes; while, on the other hand, whenever there is no blocking of the normal openings of the posterior sinuses, this region can, I believe, be ruled out as a causative factor in the neuritis. The following is quoted from Stark, who expresses well the thought I wish to convey: "From a nasal standpoint we must not expect to find the common symptoms of sinus infection, pus, polypus, history of nasal discharge, etc., as we are dealing with a closed sinus; otherwise we should not have pressure. * * * The deflected septum and middle turbinate tightly pressed against the lateral wall should always be suspected."

It is not always possible to determine the health or disease of a tonsil. Special attention is given to any enlargement of the cervical glands below the angle of the jaw. Congestion or swelling about the soft palate and pillars usually indicates infection. The tonsil is usually probed and pulled into the throat to determine its size and general appearance.

The teeth come next and it is right here that snags are usually struck. The patient as a rule has a good dentist and resents fault being found with the teeth. The patient's dentist was formerly asked to look the teeth over for possible infection but these reports were usually valueless. Dentists as a class try to preserve many teeth that undoubtedly need extraction. Bridge work, capped and pivot teeth are especially liable to harbor infection. If the teeth are suspicious or if no other probable focus has been found, an expert odontologist is asked to look them over. He films them, notes the fillings and reports fully on every tooth that is at all suspicious, devitalized ones especially. By this careful method of investigation the teeth are now found to be one of the most common foci of infection. Dr. Rice has kindly consented to discuss this phase of the subject. The paper he presented before the Triological Society in Montreal was a masterpiece. It stated the subject of focal infection from teeth in exactly the way I would like to have every ophthalmologist and rhinologist view it.

Now as to the sinuses, the patient is referred to a radiologist, who usually takes two anteroposterior and one lateral plate of the sinuses and one of the optic canals. Should the pituitary be under suspicion additional plates may be needed of the sella, but as a rule this region is well shown on the one lateral place. Whenever the sinuses are negative the teeth and tonsils should be suspected. Tonsils at all suspicious, with teeth and sinuses negative, should be removed. The diagnosis many times must be made largely by exclusion. In the hospital cases physical and neurological examinations are made a part of the routine as well as a Wassermann. In private practice this is done

in all doubtful cases. One should consider in turn blood, urine, hysteria, pellagra, lues, tobacco, alcohol, lead, arsenic, quinin, etc. Pituitary disease, brain tumor and multiple sclerosis, while not usually producing such *sudden* loss of vision, must always be borne in mind. As a matter of hospital routine the various cranial nerves and lobes of the brain are tested. Not a few cases with brain tumor have had various nasal operations before they were correctly diagnosed. It is usually possible to accumulate all this data within forty-eight hours. It is always best to look at these cases in a quiet, sane manner. The patients are usually greatly disturbed over their loss of vision, so let us not become undully excited, as most of them would recover if left alone. Should some definite focus be found, this should be eliminated unless the patient is already much below par. Should he be convalescing from influenza or have some other debilitating disease, there might be danger of losing his life. Three or four of my patients were such poor surgical risks that it was necessary to wait some weeks. Unless some definite focus is found it is usually best to treat the case. The intestinal tract should be kept healthy and the patient put in the best possible condition to combat his infection, as in many instances retrobulbar neuritis only comes when his resistance has been impaired.

In some cases the drainage and ventilation of the posterior sinuses is impaired possibly by negative pressure. This would favor the migration of bacteria and toxins from the blood stream to the optic nerve, a condition similar to what usually exists in the tympanum in Bell's palsy. In such cases it has been my practice to use a nasal spray of 10 per cent argyrol, and adrenalin about the middle turbinate every three or four hours. Hot saline irrigation, tampons of argyrol, and hot mineral oils all tend to assist in the establishment of the ventilation of the sinuses. In about 15 per cent of these cases where there is reason to fear optic atrophy, removal of the middle turbinate and opening of the sphenoid and posterior ethmoid cell is advocated.

I have in my records 60 unreported cases with some type of optic nerve disturbances. The detailed report of 33 of these appears in the Transactions of the American Laryngological Association for 1926. These cases will, I believe, substantiate my contention that most cases recover by simply removing the focus of infection.

The tonsils appeared to be the only focus in 17, the teeth in 14, and the antrum in two. Both teeth and tonsils appeared infected in four cases, while in six it was found in teeth, tonsils and antrum. In but six cases were the ethmoids involved, and even in these there was also infected teeth, tonsils or antrum.

The posterior sinuses were opened in but nine of these cases. In two recovery would probably have taken place fully as speedily had the sphenoids not been opened, while in five it did not benefit. Thus, in but two out of the 60 cases was it of any special value.

A definite focus was found in 53, while in three it was thought to be systemic, diabetes in one, hyperthyroidism in one, postscarlatinal in one. Two cases were negative except for marked blocking by hypertrophied middle turbinates. A focus was not discovered in one case, while another had an acoustic neuroma.

The surgical elimination of the focus was followed by normal vision in 23, marked improvement in 12, slight in five, none in six. In seven refusal to have it removed was folowed by no improvement in six and normal vision in one. Three cases recovered under treatment, while one showed marked improvement. A fifth case (the diabetic) did not improve. The patient with the acoustic neuroma died, as did one with carcinoma of the sphenoid.

Thus, teeth and tonsils appeared to be the foci in about 70 per cent of these cases, while the ethmoids were involved in but 10 per cent, so there would seem little excuse for longer considering ethmoids and sphenoids the all-important factors in optic nerve disturbances. They rarely require exenteration. The important thing today in rhinological surgery, as in all other branches, is to remove the focus.

SUMMARY.

It is usually possible to find the focus of infection and by eliminating it, before the nerve is damaged, to obtain prompt recovery. Nasal obstruction when well marked should be removed. As the posterior sinuses are rarely infected it is unnecessary to open them except in a few desperate cases. Papers advocating exenteration of the ethmoids and sphenoids, even when they show no definite infection, are to be condemned. The benefit following the opening of the sinuses would naturally occur about that time, although some of it might be due to the depletion of the structures adjacent to the nerve. As sufficient pathology was not found in the sinuses to account for these optic nerve disturbances more distant foci were sought. The removal of infected teeth and tonsils soon proved to be the most effective method of treatment. Infection is now believed to come from either some systemic condition, as influenza or intestinal toxemia, or from some definite focus in teeth, tonsils or sinuses. This infection travels by way of the blood stream and produces a neuritis of the optic nerve. Eliminate it from the blood stream and the neuritis subsides.

The prognosis is dependent upon the duration and extent of the loss of vision, the condition of the fundus, the virulence of the infection and the size of the optic canals.

About 85 per cent of the cases would recover under conservative treatment, such as local medication and the removal of some definite focus. These cases are usually taken too seriously. Extreme measures are instituted where little or no treatment would suffice, and while recovery may take place in spite of, rather than because of, radical operative procedures, the patient is submitted to a needless risk.

In the remaining 15 per cent where we are contending with a severe neuritis with marked or complete loss of vision, it is usually advisable, in addition to the removal of the focus, to also remove the middle turbinate and open the posterior sinuses.

Teeth should be studied by an expert odontologist, devitalized ones especially.

Negative pressure in the sphenoid would favor the migration of bacteria and toxins from the blood stream to the optic nerve, a condition similar to that which takes place in the tympanum in Bell's palsy.

A definite focus was found in 53 of the 60 unreported cases. The tonsils appeared to be the only focus in 17, the teeth in 14, and the antrum in two. Teeth and tonsils either alone or combined was considered to be the focus in 70 per cent. In but six cases were the ethmoids involved and even in these there was also infected teeth, tonsils or antrum. Th frontal and sphenoid were each involved but twice. The removal of the focus was followed by normal vision in 50 per cent and marked improvement in 25 per cent. Much better results would have been obtained had the focus been removed earlier.

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390 Commonwealth Avenue.

RHINOLITHIASIS—REPORT OF TWO CASES.*

DR. A. LOBELL, New York City.

The pathogenesis of rhinolithiasis is that of stone formation in other parts of the body, such as the gall bladder, the kidney, the salivary glands, etc. Bacteria, leucocytes and other cells of the surrounding area form a nucleus around which the salts of magnesium, calcium, etc., are deposited until a visible concretion is formed. This may be termed a rhinolith of endogenous origin. It is possible for an extraneous foreign body within the nose to become the nucleus of a rhinolith in a similar manner.

Judging from the frequent accumulation of secretions and pus within the nasal cavities, one would expect rhinoliths to be encountered more frequently in practice. The rarity of its appearance might, however, only be apparent, for it is possible that many a nucleus or small rhinolith is extruded from the nose by blowing before it has a chance to become imbedded.

Diagnosis: The history and physical examination without the submission of a specimen for examination to the laboratory are not conclusive for exact diagnosis. Syphilis even without a positive Wassermann suggests itself from the beginning because the hard, bony structure resembles a sequestrum in all respects. The X-ray is of no value in the differential diagnosis, because the rays are obstructed in each instance by identical chemical elements. The Roentgenologist can only report bone necrosis. If a metallic substance should happen to be the nucleus of the rhinolith the X-ray would of course be of immense value in the diagnosis of such a case.

Treatment: In those instances where the rhinolith is fairly large, deeply imbedded and of long duration, it is well to be prepared for some difficulty in removing it. Several problems will present themselves. These are: *1.* anesthesia; *2.* bleeding; *3.* complete removal.

1. The best patient will not tolerate the manipulation incident in its removal by the mere swabbing of the surface mucosa with a local anesthetic. It is preferable to infiltrate the mucoperiosteum with novocain, 0.5 to 1 per cent. If this is not feasible on account of anatomical inaccessibility, one may resort to blocking, as for a submucous resection. Nervous patients may require a general anesthetic.

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2. While there is no danger of any serious hemorrhage during the operation, nevertheless the bleeding is continuous and sufficiently profuse to cause considerable annoyance. One must be prepared for this and if the patient is warned ahead of time the operation will not have to be abandoned at the first attempt. The bleeding is excessive on account of the new blood vessels which permeate the exuberant granulations in which the rhinolith is imbedded.

3. From the examination by the probe and the X-ray plate one should obtain a comprehensive idea of the size of the stone. It is

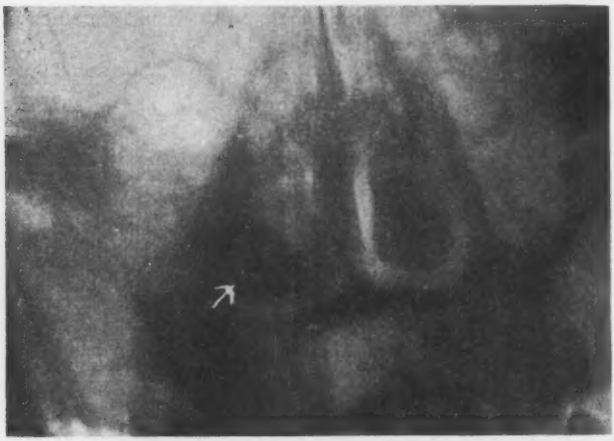

Fig. 1. Roentgenogram. Anteroposterior view. Arrow pointing to location of rhinolith, right nasal fossa.

unlikely to be removed in one piece when it is imbedded. The grasping forceps crushes it easily because it is brittle in its exposed portion. The deeper parts should be freed from the surrounding soft tissues by dissection. It will then be a simple matter to grasp the stone by means of a suitable forceps and remove it slowly after rocking it gently from its bed, not unlike the dentist extracts a tooth. Care is necessary to prevent loose fragments to remain floating in the nasal cavity because of the possibility of such pieces to drop into the larynx and bronchial tree.

Case 1: In the month of April, 1926, a woman presented herself at the Manhattan Eye, Ear and Throat Hospital on the service of Dr. John E. MacKenty under the care of the writer. She was

married and gave a negative luetic history. Her chief complaint was that for the last three years she had a foul ichorous discharge from the right nostril, nasal obstruction and supraorbital pain on the same side.

Examination: The right nasal fossa was filled with a thick sanguinopurulent, malodorous discharge. After clearing away this material there was seen an elevated mass of granulation tissue surrounding a grayish white body. A probe revealed that the whitish portion was of bony consistency and the soft tissue bled very easily to the

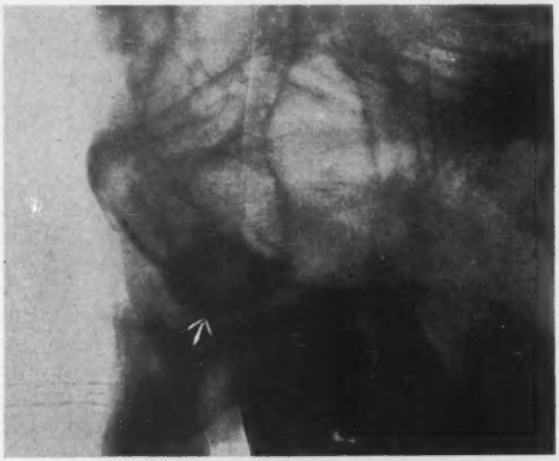

Fig. 2. Roentgenogram: Bilateral view. Arrow pointing to location of rhinolith in floor of right nasal fossa.

touch. The probe could be passed into the right antrum in this region, showing that the antromeatal wall was destroyed in this neighborhood. The inferior turbinate was undermined in its middle portion by this mass of supposed dead bone. A blood Wassermann was negative. The X-ray report was bone necrosis in the floor of the right fossa. The right antrum had a severe involvement, the left was clear (Fig. 1-2). A small piece was removed for examination and the report was rhinolith.

Operation: Under local anesthesia with novocain infiltration the stone was removed (Fig. 3). It was found deeply imbedded, encroaching on the antromeatal wall and lying exposed in the antrum

in part. The bleeding was profuse. The stone measured, roughly, 1x¾x½-in. After its removal there was a large opening in the antral wall, through which irrigations were carried out. The antrum cleared up within several weeks and the supraorbital pain disappeared completely.

Case 2: Male, age 32 years, married; luetic infection denied. The nose was fractured in childhood, resulting in a deformity. He complained of nasal obstruction on the left side, a foul discharge and occasional epistaxis. These symptoms were intermittent for the last five years.

Fig. 3. Rhinolith, small pieces missing. Actual size.

Examination: Saddle nose, resembling the luetic type. Sinking of the cartilaginous portion of the septum. A blood-tinged, purulent discharge from the left side. Half-way in the fossa, on the floor of the nose, there was a white ivory-like body surrounded by granulation tissue. The Wassermann was negative. The X-ray showed bone necrosis. A small piece was examined and the report was rhinolith. Unfortunately, this patient refused the operation for its removal.

1420 Grand Concourse.

A CASE OF ANTRAL SINUSITIS COMPLICATED BY RADIUM POISONING.*

Dr. Frederick B. Flinn, New York.

Radium has never been classified among industrial hazards, probably on account of its high value and the small quantity in existence. That precaution must be taken in handling radium and in its medicinal use has long been recognized. It is estimated that the entire world's production to date falls somewhere between 200 and 220 grams, approximately 100 grams of which are in use in North America. It finds its chief commercial use in the painting of clock and watch dials or similar instruments. Mesothorium has replaced radium both for commercial and medicinal purposes in some instances. The radioactivity of a substance is expressed commercially in terms of radium. When one speaks of one milligram of mesothorium—which is more active than radium, weight for weight—one means the amount of mesothorium that will give off the same amount of emanation as one milligram of radium would give off.

In the radium series some of the products following radium C are long-lived and gradually form products giving off alpha rays. This is important because recently Lind has shown that the alpha rays are ten or fifteen times as powerful as at one time estimated. In the thorium series, on the other hand, the products which follow emanations are comparatively short-lived and hence if the disintegration products from the emanations have at all contributed to the illness of those exposed, it would seem that the radium products are the ones responsible.

There are two main exposures in the industrial production and use of radium:

1. Exposure to the emanation which may result in radioactive deposits in the body; or to external burns when the material is handled in a careless manner.

2. The ingestion of the radioactive material either by the inhalation of the dust, or by the actual ingestion of the material by mouth through ignorance or willfulness on the part of the employees.

The first hazard is well understood and the measures for protection known. The emanation is a gas and calls for good ventilation of the

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premises to prevent any accumulation. Furthermore, all radioactive solutions should be kept under hoods with good draughts. Lead protection screens or thick lead glass should be placed around the measuring apparatus as well as between the workers and apparatus. There should be a strict rule against the handling of tubes, needles, etc., with the fingers when they are being filled with radium salts. Carelessness in this matter may result in local affections. I have seen cases where long continued exposures of the fingers to radium has produced radium keratosis. This develops a more virulent lesion—possibly a squamous epithelioma. These lesions are apparently of low virulence, as it has been noted in one case where the keratosis had been present for seven or eight years that it did not grow and was not sensitive except when radium was brought in contact with the lesions.

Emanations from the body act on the hemopoietic system of the body and the intensity of their action depends on the quantity and length of the exposure. Certain facts stand out in the blood changes from gamma ray exposures in the work of the Medical Research Council of London: *1*. The rapid and profound destruction of lymphocytes; *2*. the rapid destruction of red corpuscles under prolonged exposure; *3*. the sign of blood destruction and lack of regeneration in the bone marrow. In the case of the leucocytes a progressive leucopenia occurs, essentially due to a great destruction of lymphocytes.

The number of polynuclear cells is increased for a time, but with a prolonged exposure they also suffer destruction. It has been considered by some investigators that the red cells are more resistant to the effects of radium than the white cells. The London Radium Institute after a study by Mottram came to the conclusion that a consistent white count below 6,000 among radium workers should be considered as indicative of an undue exposure and the person removed from the work until the blood condition became normal again. This measure is apparently sufficient unless the exposure has been too long or severe, resulting in a marked leucopenia with white counts below 1,000 in fatal cases. Personally, I feel that if the blood picture is indicative of an undue radium exposure, the worker should not return to the work again, but should be assigned to other duties.

I have examined a large number of radium workers and have come to the conclusion that mere dependence on the white count alone is not sufficient to indicate whether or not a person is receiving an undue exposure. The situation is further complicated by a study of the blood counts of numerous non-industrial workers, which empha-

sizes the fact that the so-called normal range means little in a survey of this nature. Furthermore, it is evident from a study of some of the fatal cases that the blood picture may remain more or less normal until some infection arises or perhaps some change in metabolism and this results in blood changes that are considered typical of radium exposure. Most of the cases of death which have been reported as due to radium were due to some other cause, radium being a secondary agent, in that it had lowered the resistance of the body. In making this remark we are confining ourselves to cases of small exposure over a long period of time.

Recently the danger from radium ingestion has received attention —principally among radium dial painters. This work consists in applying radioactive material to the hands and numerals of the dials. Contrary to the popular notion, the material used does not contain phosphorus. It consists of pure zinc sulphide which has been prepared by repeated roastings and reprecipitations. To this zinc sulphide is added approximately .05 per cent of cadmiun, .001 of copper and .0002 per cent of manganese. To the emulsion of this mixture is added a soluble radium salt which is then precipitated out as a sulphate by the alkaline sulphate. Barium sulphate is one hundred times more soluble than radium sulphate. The radium content of the luminous paint varies from approximately .7 mgs. to 3 and 4 mgs. per 100 grams of zinc sulphide. This luminous material is mixed with gum arabic and thinned out with distilled water. Only a small amount is mixed at one time in a 10 c.c. porcelain crucible with a glass rod. It is applied by means of a camel hair brush by dotting rather than drawing the brush over the numeral. This dotting procedure requires a well-pointed brush, and it is for this reason that the painters were accustomed to pointing the brush between the lips in order to speed up the work.

It is suggested that this habit of pointing the brush between the lips is the cause of the so-called radium necrosis of the jaw. Just how much paint a girl might ingest with this procedure is a question. We have had girls point their brushes on a clean cloth for a week and have then determined the amount of paint on the cloth, having also kept a record of the weight of the material that each girl used. We found that it might be possible for the careless girl to ingest approximately half a gram of the paint for each 10 grams of the paint that she used. It is hard to say how much paint a girl would use in a day because it depends on the type of work being done. In the plant in which this test was being made, the operator used about 10 grams a week. It was found that this half gram of paint con-

tained .0072 mgs. of radium. In other words the operator might in some cases have passing through her alimentary tract .0072 mgs. of radium each week. Of course only a very small portion of this radium would be present at any one time. And yet, considering the feeding experiments in which charcoal is used to indicate the time it takes material to pass through the body from the time it is ingested and the waste products excreted, one can estimate that there is present in the tract 15 or 20 micrograms continuously. Experiments with animals indicated that practically all of the ingested material is excreted. There is some absorption of radioactive material into the body, where it is finally deposited in the bones in the same way that lead is. If the feeding of the luminous paint to the animals is discontinued there will in course of time be a complete elimination of the radium. In the case of an intravenous injection of radium salts we have found that approximately 60 per cent of the salt will be eliminated in ten days and that after that the elimination is very slow, often only 1 per cent a month.

In June, 1926, a case was brought to my office by a dentist because he felt that the condition of the jaw might be explained by a previous industrial exposure. The young woman had painted dials for fourteen months in 1921-1922. She had had meningitis three months previous to beginning dial painting and received the serum treatment. About two years ago she tripped and snapped her femur without actually falling to the floor. Considerable trouble was experienced with this fracture because of improper setting, which necessitated refracturing and resetting. About a year ago she was treated for pyorrhea. Ten weeks previous to her visit to my office the two lower first molars and a first bicuspid had been extracted as they were loose and the bone exposed in the left molar region. Following this extraction a swelling developed over the body and ramus of the left mandible, extending over the face up to the temporal region. The swelling continued and was of moderate size when I first saw her, apparently on account of a trismus involvement, and she could open her mouth only about half a centimeter. The soft tissue in the lower left first molar and second bicuspid region was inflamed and slightly tender. The buccal alveolar plate of the bone was exposed and necrotic. It had a dull, grayish appearance on account of a very moderate discharge of pus. The exposed bone was solid. The second molar was quite loose and was extracted within a few days after our examination. The right side did not present so advanced a pathological condition as the left side, but the tooth socket had not healed. One or two of the teeth were denuded

and slightly loose. The condition was diagnosed as necrosis of the mandible.

An X-ray of the jaw showed an area of apparent bone destruction on the left in the region of the bicuspids, which were missing. The area appeared very irregular, but no shadows could be seen suggesting bone sequestra. There was dense shadow anterior to the first molar suggesting calcification. There appears to be an area of absorption about the roots of the incisors. On the right the mandible appeared rather mottled in density just below the roots of the incisors. There was also a small area of decreased density below the roots of the second molar, which appeared to be a small area of absorption.

An electroscopic test was made both by the emanation and the gamma ray method. The emanation test consists in having the patient breathe into a Douglas bag and then in drawing the expired air into an alpha ray electroscope. This method is of value in confirming the presence of radium. It is not a quantitative determination, because if radium is present as a sulphate only 5 per cent of the rays can escape from the salt. On the other hand, if it is in a colloidal condition 75 per cent can escape. The gamma ray test consists in placing the electroscope close to the body and making the customary readings. This will give a good estimation of the amount of radioactive material within a radius of 30 inches of the instrument. It was estimated by this method that the body of the patient contained 100 micrograms of radioactive material. We may say that we have injected known amounts of radium salts into the bodies of animals and have been able to check these amounts by means of the gamma ray tests.

The patient was sent to the hospital for observation and treatment. Reasoning from the action of radium, we decided to give her 5 grams of ammonium chlorid each day to see if we could assist in the elimination of the radium. We were unable to carry on this treatment for more than four days, because of the objections of the patient. This treatment has never before been tried for radium poisoning, but there are indications that it might have been successful if it could have been continued. As it was, we were able to cut down the radium deposit in the body to 86 micrograms. Of course this was only an indication of what might have been expected.

On admission to the hospital, the patient's blood showed a hemoglobin of 35 per cent, R. B. C. 3,100,000, W. B. C. 6,100, and a normal differential count, the polymorphonuclears being 64 per cent, and the lymphocytes 26 per cent. There was a slight aniocytosis and

poikilocytosis, polychromatophilia. Blood pressure 105/65. Wassermann negative. Poor menstrual history. A blood transfusion was given a week after her admission to the hospital, with apparently good results, for in the latter part of August her hemoglobin had risen to 55 per cent and the red count stood at 3,600,000.

The latter part of July the patient complained that pus was being discharged from a tooth in the upper jaw. An X-ray showed diffused clouding in the region of the nasal accessory sinus, the condition being most marked over the right front antrum and ethmoids. The antrum was punctured on July 27 and a large amount of very foul pus was washed out. Strep. viridans was found to predominate in the discharge. Her antrum condition did not improve and a radical operation was performed the first week in September. An examination of her blood after the operation showed that the hemoglobin had dropped to 35 per cent and the R. B. C. to 1,600,000. She was given two blood transfusions two weeks apart and on Oct. 17 her hemoglobin was 52 and R. B. C. 3,730,000. The gums are being treated with mercurochrome and the mouth irrigated with 50 per cent of hydrogen peroxid.

The patient is being kept on high nucleic acid diet. There is some improvement in her general condition at the present time, and she can open her mouth about 1½ c.m. But indications of anemia are present. There is still a slight discharge of pus from a sinus in the lower left jaw. A bacteriological examination showed staph. aureus +++; strep. hemolyticus, +; B. pyocyaneus, ++.

Jaw necrosis has occured in six cases among radium dial painters that we know of, all of whom had been employed in the same factory during the same period of time. Five of these cases were fatal, while the sixth is apparently recovering from her exposure. An X-ray of her jaw shows that the necrotic condition is still present. An electroscopic test indicated she still retains 10 micrograms of radium in her body two years after her treatment first began. Her dentist diagnosed her condition as a low grade infection of the mandible. The necrosis in these cases is said to differ from phosphorus poisoning or ordinary necrosis or typical osteomyelitis only in the number of areas involved and the remote location of the areas from one another. In the later stages of the diseases these necrotic areas presnt a liquifaction more or less of a gelatinous character rather than a bony disintegration. Unfortunately the cases were complicated by diabetic and syphilitic conditions. Vincent organisms were found to be present.

I have examined practically all of the girls who are now working at dial painting in this country and have made inquiries in most of

the European countries, and had yet, up to June, 1926, to find another case that had been reported among the three or four thousand girls that are working or had worked in this industry outside of the one plant mentioned. The examination included the regular blood count, X-ray picture and electroscopic observation, and an inspection of the condition of the mouth. Under the circumstances I was forced to the conclusion that the necrosis among the six cases reported was due to some peculiar condition in that one plant and probably to some low grade of infection. This seemed quite probable because some of the girls admitted that they were in the habit now and then of borrowing one another's brushes. I have been in close touch with the industry during the past two years and it is probable that any other case other than those mentioned in the Orange Plant would have been brought to my attention. The case under discussion made its first appearance last June and has been followed by a second case during the last month. Both of these cases occurred in the same plant in Connecticutt. In view of the evidence in front of me I feel that radium is partially if not the primary cause of the pathologic condition described. I am impressed by the fact that in all of these cases radium has been present in the body, while I have been unable to find it in the persons of but three or four girls now working. I had not examined these three or four girls previous to the middle of October, 1926.

It has been suggested that particles of radioactive substances may get into the bone through the open spaces in the cancellous structure connected with pyorrhea. So far my observations do not reveal any more radium deposit in the jawbone than there is in the other bones of the body. Consequently if radium is to be blamed for this condition it must be due either to a general systemic condition or to the exposure of minute quantities of radioactive material passing through the mouth or lodging temporarily in the teeth. Animals did not reveal any irritation of the gums when they were painted daily for six months. If the presence of small amounts of radium in the body will produce such profound systemic changes as to lower the body's resistance to the extent indicated in these cases, one cannot but view with alarm the intravenous injection or internal administration of radioactive material. If radium has affected anyone who has had such treatments, the action of the radium may have been masked by the other complications.

The condition of the jaw under discussion has been compared to phosphorus necrosis. As is well known now, it was not phosphorus that caused necrosis, but bacterial infection. It has been shown by

Thorpe that as much as 0.37 per cent of the weight of a tooth could be destroyed by phosphorous fumes in twelve hours in case of decayed teeth. What phosphorus did was to cause an irritation which opened channels for the bacterial infection.

Lazarus Barlow in discussing the treatment of some thirty-six cases of malignant disease with a large amount of radium, says that the degree of injury to the skin seemed to be definitely influenced by the local vascular supply. In situations where the nutrition of the skin was poor on account of close proximity to affected areas, a short exposure which usually does no harm has often caused vesication. The effect of the radiation in such cases has been sometimes to hasten ulceration, and an additional factor influencing this result is the effect of radiation on sepsis. There seems no doubt that if a growth is already infected, radiation produces a favorable condition for the propagation of bacteria. This is probably due to a lowering of the resistance of the tissue.

Bone necrosis is known where carcinoma has been treated with radium, especially where glass seeds are used, as the glass is not sufficient to filter out the beta rays. Radium necrosis may not occur for a period of months, or even years, after exposure to radium. Porosity is first noted, gradually developing into complete disintegration and sequestration. It is this distinction that makes us feel that the cases of jaw necrosis among radium dial painters was due to a bacterial invasion, as indicated by the gelatinous nature of the bone in most of the cases. There is a bare possibility that an irritation of the gums is caused by the sulphides in the paint being oxidized because of their finely divided condition and the action of the fluids in the mouth. We are familiar with necrosis of the gums caused by copper sulphate. The argument against this theory is that most of the cases on record did not appear for two or three years after the patient had withdrawn from the occupation.

This case showed a gradual improvement until the first part of December when a heavy cold developed. From that time until her death the latter part of January, the patient became progressively worse. X-ray pictures showed no bone regeneration. The sinuses were full of green gangrenous material. The eyesight became affected. Death was probably due to a general toxemia. A blood examination showed 4,000,000 R. B. C., 6,000 W. B. C., with a normal differential count. Hemoglobin 50 per cent. The autopsy revealed no lesions other than that the bone marrow presented the same general condition as is found in benzol poisoning. An electroscopic test made the latter part of October showed that the radio-

active deposit in her body had been reduced from 100 to 75 micrograms during five months.

Whether the small amounts of radium present in the bones is sufficient to cause systemic changes has been questioned. There is not enough evidence at this time to permit one to take a definite stand. I can say that if 50 milligrams of the luminous paint, containing .00072 mg. of radium and mixed with 1 gram of bone ash, is placed on a dental film for four weeks it will cause a definite fogging of the film. This being true, it may be that a constant exposure of the hemopoietic system to these small quantities of radium may in time result in a lowering of the body resistance to infection. Radium being deposited in the bone may bombard the marrow continuously with the powerful alpha and beta rays. Coolidge in discussing the powerful effect of his cathode rays and their destructive properties, called attention to the well known fact that they are similar to or the same as the beta rays of radium. We have already called attention to Lind's findings on the alpha ray.

It may also explain why the blood examination does not always give warning until the damage is past repair, an explanation being a continuous stimulation of the blood-forming mechanism to the extent that it has used up its powers of regeneration. This is indicated by the fact that we have seen an actual increase of red and white cells during the early part of an exposure.

Enough has been said to indicate the necessity for a study of the physiological effects of continuous exposure to small amounts of radium over a period of years.

MUCOCELE OF THE FRONTAL SINUS, WITH THE REPORT OF A CASE.*

DR. W. T. GARRETSON, Detroit.

The comparative rarity of this disease has stimulated the author to report a case. Sir St. Clair Thomson and Dr. Chas. P. M'Illraith in 1923[1] reported that only 100 cases of this disease had been reported to that date. Dabney, of Washington,[2] in 1921 reviewed the literature and listed 58 cases. It remained to A. Logan Turner, of Edinburgh, and Gerber, of Germany, to chiefly elucidate the condition and place the information on a more scientific basis.[3]

Definition: By the term of mucocele of the tissue is meant the accumulation and retention within it of mucus and mucoid secretion with blocking of the sinus ostium, and with thinning of the bony walls of the sinus, with possible distention of one or more of its walls. When pus is present it is known as suppurative mucocele or pyocele. The occurrence of mucocele is not uncommon. Any one of the paranasal sinuses may be affected, although the sphenoid sinus is rarely so. Howorth,[3] who has seen 14 cases of mucocele of the frontal sinus and the anterior ethmoid cells, has not yet observed a single case involving the sphenoids. With the accumulation of retained fluid in the sinus the intrasinus pressure is increased and the thinnest walls tend to dilate and ultimately absorption takes place and the bony wall ultimately gives way. This usually occurs in the roof of the orbit in the region just behind the trochlear fossa or just external to it.

Clinical Picture: The chief clinical picture of mucocele of both ethmoid and frontal is orbital in character. The patient usually goes to an ophthalmic surgeon in the first instance as frequently the first thing to be noticed is the swelling at the inner and upper angle of the orbit, which feels hard and bony. If there is much bony absorption a parchment-like crackle may be noted on palpation. When the dilated or expanded bony walls have been absorbed the swelling is definitely elastic or fluctuant. There is no tenderness or pain on manipulation. The skin is not adherent to the underlying structures and is not altered in character and moves freely over the subcutaneous tissues. Pressure over the swelling may not alter its size. The tumor-like

*Department of Ophthalmology and Otolaryngology, Henry Ford Hospital.

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mass may remain practically stationary for years. However, when the bony covering has been absorbed the condition progresses more rapidly and symptoms of pressure on the orbital contents may be noted.

Classical Picture of This Disease: In the classical picture of this disease the eyeball is displaced downward, forward and outward. If the mucocele is of a large size, proptosis may be marked. Diplopia is manifested as soon as much pressure is made against the orbital contents. In certain cases a high degree of displacement occurs without diplopia. In others, diplopia is one of the earliest signs. The movements of the eyeball are usually unaffected as the tumor mass is outside of the orbital contents and, in fact, under the periosteum of the roof of the orbit. If the mucocele is ethmoidal the swelling is usually lower down and may displace the lacrimal apparatus. This often leads to mistakes in diagnosis. The chief point of difference, however, is the presence of pus coming from the canaliculus when pressure is made over the lacrimal sac or possibly the presence of pus or mucopus in the nose as a result of the discharge through the nasolacrimal duct. In the latter case the swelling is usually below the internal canthal ligament of the eyelids, while in case of mucocele and frontal sinus the swelling is above this ligament.

Etiology and Pathology: The onset is so gradual and the progress so slow and painless that a definite cause for this is difficult to assign in any case. A. Logan Turner,[4] of Edinburgh, is of the opinion that we are justified in thinking that a persistent catarrh may be the reason for the changes which take place in the sinus walls about the nasal frontal duct, causing obstruction to the outlet with the resultant accumulation of mucus content in the cavity and a gradual dilatation and absorption of the bony walls. Killian (quoted by Walter G. Howorth[3]) and others have suggested the possibility of previous injury to the region of the nasofrontal duct. In A. Logan Turner's[4] 14 cases no evidence of previous injury could be found in a single case. In general two possible explanations may be offered as to the cause of this condition: *1.* Inflammatory. *2.* Traumatic.

However, certain anatomical variations in the relation of the anterior ethmoid cells at the nasofrontal duct may be noted. The anterior ethmoid cells may almost surround the nasofrontal duct and in some cases extend anterior to it, and even bulge into the floor of the frontal sinus. Such abnormalities narrow the nasofrontal duct and in case of low grade inflammation may give rise to plastic inflammation of the mucous membrane lining the duct and cause constant obstruction.

Symptoms: Symptoms may be entirely wanting until the external swelling appears. Slight alteration of the visual axis as a result of displacement of the eyeball and orbital contents may give rise to diplopia. Sometimes headaches are complained of and in others only a brow ache, and in others there may be a generalized uneasiness or headache.

Clinical Diagnosis: In some cases confusion in diagnosis may occur as a result of a superimposing of an acute infection of the contents of the mucocele. In others the differential diagnosis must be made from cystic dilatation of the lacrimal sac. The latter is more frequently confused with mucocele originating in the anterior ethmoid cells. If it is kept in mind that pressure on the lacrimal sac will usually express mucopurulent fluid through the nasolacrimal duct into the nose or through the canaliculus and the presence of the tumor above the internal canthal ligament in cases of ethmoid mucocele and the failure of mucopurulent material to be expressed from the canaliculus the diagnosis should be definite. More difficulty is presented by tumor of the frontal sinus and of the orbit, such as fibrosarcoma, osteomata, dermoid cyst and meningocele, which may occur in this region. Roentgenograms are of much help in tumor and osteomata. Dermoid cysts more frequently occur at the outer canthus but frequently occur at the inner. They are frequently subperiosteal and palpation reveals a depression in the bone beneath the tumor mass; they are displaced with difficulty as they are bound down by the periosteum. They are usually present from birth or shortly afterward and gradually increase in size. In meningoceles they may occur in this region, but, like dermoids, are congenital. The bone is not expanded and X-rays of sinuses show no alteration in the density. The age factor in the differentiation of these conditions is also of considerable importance, in that mucoceles occur on an average between 33 to 85, whereas dermoids and meningoceles are noted much earlier.

The Duration: The duration of the condition cannot be definitely stated in any individual case unless it can be traced to an accident, such as a skull fracture or broken nose. Study of a great many cases by the author has given him the impression that the duration in most cases is a good many years and in certain cases as many as twenty. The average occurrence in the literature is about 60, but the age varies from 40 to 85. The author's case is the youngest which he has been able to find. Patient's age is 33 years.

I wish to submit the history of the following case: Male, age 33 years. He first came to the clinic in July, 1923, complaining of a

protrusion of the left eye, with double vision. There was a history of having been struck over the left eye by a baseball a number of years ago. He did not notice the trouble until 1916, when diplopia was first noticed. Since that time he has noted a gradual protrusion of the left eyeball with tendency of the eyeball to turn downward and outward. He has never noted pain or headache and has always had free ventilatoin on both sides, but colds have been frequent, but between times has noted no unusual nasal discharge. The family and past history are negative.

Physical Examination is essentially negative, excepting for a small hydrocele and the protrusion of the left eye. On examination the left eye protrudes markedly downward, outward and forward. The movements are normal in all directions. On palpation of the upper part of the orbit a definite tumor mass, fluctuant in character, projects from the roof on the inner side of the orbit. The bony floor of the frontal sinus has been absorbed. Vision in the left eye is 6/60 and ophthalmoscopic examination shows considerable optic atrophy with marked engorgement and tortuosity of the retineal veins. Both maxillary sinuses and the right frontal illuminate well. Anterior and posterior rhinoscopy show no abnormalities. Ears show nothing abnormal. Blood Wassermann, plain and cholesterinized, antigen, is negative. Urine examination shows no abnormalities. The X-rays show an indefinite crescent-shaped shadow in the upper and inner portion of the orbit. On the basis of this history, the absence of pain, the gradual slow onset and the gradual progress of the disease with the absorption of the floor of the frontal sinus, with projecting tumor, with the X-ray findings a diagnosis of mucocele of the left frontal sinus was made and on this diagnosis an operation was advised. Patient consented to operation on Oct. 1, 1923, at which time a modified Killian operation was done. The operative findings showed a large mucocele completely filling the frontal sinus and fully one-third of the orbital cavity. This, however, was entirely subperiosteal as far as orbital content was concerned. The levator palpebrae superioris was not involved. The roof of the frontal sinus was absorbed, with exposure of the dura of the anterior fossa to an area of the dura of the anterior fossa to an area of about ¾x½ inches in diameter. There was complete destruction of the floor of the frontal sinus. It was noted that the anterior ethmoid cells were normal, but considerably compressed. The os planum was bulging inward. At the time of the operation the os planum and ethmoid cells, anterior group, were leveled off to the level of the bony floor of the middle fossa. The middle turbinate and part of the nasal

process of the superior maxilla were likewise removed so as to give free access from the nose to the frontal sinus. No attempt was made to remove the mucous membrane from the lining of the frontal sinus and the orbital projection. Patient was seen eleven days after discharge from the hospital, at which time only a slight nasal discharge was noted. The proptosis had entirely disappeared. There was only occasional diplopia.

Patient returns on Jan. 19, 1925, one and one-half years later, in answer to a recall letter. Examintion shows there is very slight suggestion of ptosis in the left eye. Eye excursions are normal in all directions. There is no evidence of diplopia. Examination of the left fundus shows slight atrophy, but previous venous engorgement is nowhere in evidence. Vision is 6/30 and is improved with plus lens in the left eye. He should have cycloplegic refraction, but owing to rush of work is unable to do this, but will return later. There is very little scar tissue present. Patient has had no unusual discharge and breathes freely through the nose. Frontal sinus cannula enters frontal sinus region freely. The cuts illustrate patient before and after operation.

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FRACTURE OF THE OUTER TABLE OF THE FRONTAL SINUS.*

DR. SIMON STEIN, New York City.

Fractures of the skull occur quite frequently and may involve the sinuses as well as any other part. Trauma to the nose particularly produces fractures which may and do extend into the sinuses. The following case is rather unique in its involvement and subsequent course:

On June 26, 1926, several boys staged a miniature battle in the streets, using "corn on cob" as implements of warfare. Patient J. S., age 32 years, was unfortunate enough to be caught in the crossfire, and was hit in the left temple. What followed can best be explained in his own words: "I felt a sudden blow on my head, which made me feel very funny. I thought I would have a swelling over the place where I was struck and was surprised when I felt for it to find that instead of a swelling I had a hole into which my finger seemed to gor for about an inch. I became very dizzy and developed a severe headache. I ran to a nearby hospital and was examined by several doctors, who seemed to be very much puzzled by my condition. They gave me some medicine and told me to go home and put cold applications on my head. I did so, and went to the hospital again the next day. They took an X-ray picture and when I returned they told me that it was nothing. My headache and dizziness persisted for over a week."

Patient at this time was treated by his family physician, Dr. M. H., who, about ten days after the accident, referred him to me. External examination showed a marked depression over the left frontal sinus area, about one-half inch below the surface. Intranasal examination showed a marked turgesence of the entire left naris, with some mucopus in the middle fossa. Transillumination was fairly good. X-ray showed a depressed fracture of the outer table of the left frontal sinus with slight clouding (Fig. 1). Fragment of bone pointing to inner table, but not reaching it on account of depth of the sinus. The left naris was cleansed very thoroughly and then the area around the frontal duct opening was adrenalinized and cocainized, followed by gentle suction. Following this treatment there was a

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freer discharge and the patient felt better. He was kept under observation for about a week, constantly improving under treatment, which consisted of an adrenalin spray and argyrol tampons at home, with cleansing, suction and baking in the office.

Since that time he is perfectly well. He has kept in touch with me and the depression seems to be filling in gradually, as evidenced by his improved appearance and the last X-ray picture that was taken (Fig. 2).

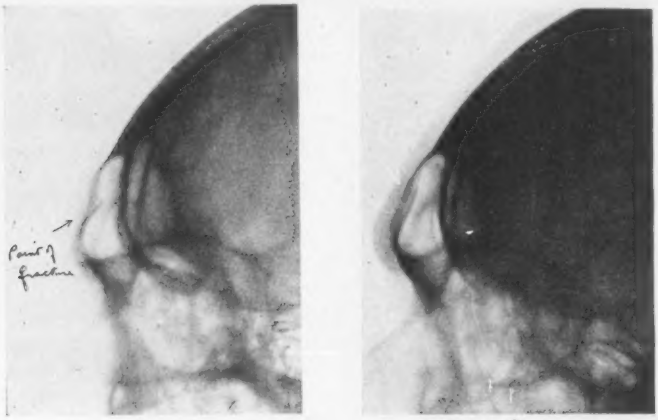

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Fig. 1. Taken one and one-half weeks after accident.

Fig. 2. Taken Oct. 21, 1926.

COMMENT.

1. The fracture itself is an unusual one; direct trauma producing a fracture, whose symptoms were comparatively slight and healing very good, with no untoward results. The marked cellular structure of the sinuses must also be taken into account.

2. The treatment was one of moderation. The nasal passages were just kept clean and drainage provided, so that there would be no infection.

3. The X-ray pictures show how well nature took care of this trauma.

225 East 19th Street.

COMBINED LUDWIG'S ANGINA, AGRANULOCYTIC ANGINA AND SEPTICEMIA.

Dr. V. K. Hart, Statesville, N. C.

These three conditions all occurred concomittantly in the same patient. Therefore the case presents a triad certainly rarely seen. The writer has never before seen even two of these pathologic conditions coexisting. The history follows:

C. C. Painful, swollen throat.

H. P. I. Five days ago had a chill, followed by a temperature. General malaise and apparent head cold. Family doctor made diagnosis of "grippe". Throat did not become sore, however, until two days later. The next day the glands on each side of her neck enlarged. Twenty-four hours after these masses appeared, which had gradually grown worse, the lids of the left eye became swollen and several circumscribed areas of injection and tenderness appeared on face. She was then sent to the hospital.

Patient states that one week before her chill the fourth finger of the right hand became red and painful. This was pronounced infected by her doctor, but subsided under treatment.

P. M. H. Appendectomy eleven years ago. Tubes and ovaries and hemorrhoids removed nine years ago. Tonsillectomy one year ago. Migraine attacks ever since second operation. Two healthy children. Normal labors.

Physical Examination. General: Robust female, age 32 years. Restless, rational, but obviously very ill. Respirations labored and rapid. Rather free diaphoresis. Temperature 103.6 (m); pulse 116.

Head: Lids left eye swollen and discolored. No edema conjunctiva. Ocular excursions good. Media clear. Eye grounds negative. There is circumscribed area of redness and tenderness right cheek and right lip (metastatic?). The throat shows diffuse injection of pharynx, soft palate and uvula. There is what is probably grayish exudate over these regions, particularly on palate. (Tonsils out, fossae clean.) Marked, diffuse, brawny swelling of the neck, marked in the infratonsillar areas, but also present beneath the chin. Limited to area of throat.

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Chest: Negative.

Abdomen: Scars of previous operations. Striae of former pregnancies. Otherwise negative.

Extremities: Reflexes present and normal. Distal phalanx of fourth finger, right hand, reddened, but no lymphangitis extending up arm. Evidently healing.

Tentative diagnosis: *1.* Ludwig's angina. *2.* Streptococcic septicemia.

Laboratory: (On previous admission two years ago, W. B. C., 9,200, and Wassermann negative.) Urine, faint trace albumin; otherwise negative. W. B. C., 1,000. Twenty-four hours later, W. B. C., A. M., 1,250 (not enough white cells on smear to count); P. M., 800. R. B. C., 4,656,000; hgb., 80 per cent. Blood culture on day of admission showed both streptococci and staphylococci. A throat culture showed streptococci and staphylococci and no Vincent's organisms.

Therapeutics and course. Bilateral incision and drainage of submaxillary area was done at once. Only serosanguineous fluid was obtained. Other than this, treatment was purely symptomatic and supportive with stress laid on fluids (by hypodermoclysis) and digitalization.

The following day an area of painful, reddened swelling appeared on the right upper arm. The swelling increased rapidly. This was opened by multiple incisions, but no frank pus obtained. The lids of the eye continued to swell, but there was no fixation of the globe, exophthalmos or choking of the disc (consequently probably not cavernous sinus thrombosis). Serum drained from the upper lid by spontaneous rupture.

Although the blood culture had not been yet reported, 15 c.c. of antistreptococcic serum (more was not immediately obtainable) were given intravenously because the patient was failing rapidly. She continued to fail and death occurred the second night (thirty-six hours after admission).

Discussion: A few words as to Ludwig's angina. This is often fatal in twenty-four to forty-eight hours. Most authorities cite the streptococcus as the usual offender.[2]

With a phlegmonous inflammation of the tissues beneath the jaw, it is perfectly possible for the infection to reach the general circulation directly or through the lymphatics. One of such processes probably accounted for her septicemia, with its consequent metastatic phenomena.

Of course, some may object to accepting one blood culture as conclusive proof of a septicemia. The course was too short to allow for another. However, the writer has previously tested the technique used in lateral sinus phlebitis. Such consistently gave the same organism (streptococcus). Control cultures on student nurses using the same media were sterile. More recently the technique has again been rigidly checked (using controls) in a case of pneumococcic septicemia complicating an acute ethmoiditis.

Therefore it is believed this was a true septicemia. Moreover, both staphylococci and streptococci were demonstrated in all transplants.

Such a septicemia probably accounts for the leukopenia, *viz.*, an overwhelming infection and paralysis of the blood-forming organs by the resultant toxins. This would suggest that the majority of agranulocytic angina patients really have a septicemia. Hunter in a recent case report and review of the literature of this condition says that the blood culture is usually negative, but streptococcus hemolyticus and bacillus pyocyaneous have been found. The streptococcus isolated in the case under discussion was nonhemolytic. He also states, "The glandular system is not involved. The course is from three to fourteen days and is usually fatal. One case has recovered and one has had a fatal recurrence in two years. Death has usually resulted from pneumonia."

Summary: There were three outstanding findings: *1.* A definite and typical Ludwig's angina. This occurred despite a previous tonsillectomy. *2.* Metastatic phenomena, suggesting a septicemia (septicemia rather than mere pyemia, because of complete systemic prostration). *3.* An angina with very low white count, the polymorphonuclears being practically absent on a stained slide (agranulocytic angina).

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Davis Hospital.

CYST OF THE LARYNX AND LOBUS AZYGOS OF LUNG. A CASE REPORT.*

DR. H. C. ROSENBERGER, Cleveland.

This interesting and unusual case is reported through the courtesy of Dr. John N. Lenker, Chief of the Otolaryngological Service of St. Luke's Hospital, to whom the patient was referred, and of Dr. H. O. Ruh, Consulting Pediatrician.

Dr. W. W. Lawrence, of Norwalk, O., whose patient is the subject of this report, has kindly submitted the following anamnesis:

"The father is of German and the mother of German-Irish descent and both are American-born. One child died at 6 years of gangrenous stomatitis, and five children are living and well.

"Baby B. was born Nov. 2, 1925, after normal labor. The color at birth was good and respiration was normal, but the cry was somewhat hoarse. About three hours after birth the child became dyspneic and cyanotic, but the dyspnea and cyanosis then and subsequently were relieved with hot compresses to the larynx. When nursing, the child seemed relieved of dyspnea.

"During the fourth week the dyspnea became rapidly worse and was not relieved with hot compresses. From birth it was noted that there was an absence of breath sounds over the right apex."

The patient was first seen by Dr. Lenker at his office, where a throat examination was attempted, causing the child to become alarmingly dyspneic and cyanotic, so that further examination was considered inadvisable and the patient was sent to St. Luke's Hospital.

The patient was admitted to the hospital at 4:30 p. m. on Dec. 5, 1925, and at that time the rectal temperature was 39.9° C. and the respirations were markedly increased to 60 per minute. Shortly after admission a Roentgenogram of the patient's chest was made and the following report submitted:

"The right chest is dull. Apparently the lung is not well inflated. The right side of the chest shows the right lung collapsed, suggesting atelectasis. The lateral view shows a widening of the upper central shadow of the chest, suggesting enlarged thymus."

The patient gradually became more dyspneic and expired at 8:20 p. m., about four hours after admission to the hospital.

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*Read before the Otolaryngological Section of the Cleveland Academy of Medicine, Friday, Oct. 29, 1926.

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A post-mortem examination was made and except as regards the larynx, lungs and pulmonary vessels, nothing abnormal was found. The report of the pathologist was as follows:

"The larynx shows a protuberance occupying the right ventricle as a rounded mass which presses the false vocal cord upward into a curve concave below, and depresses slightly the true cord. This mass measures about 3 m.m. in all diameters and appears to contain fluid. On opening it a small amount of viscid, mucoid material is found, of a clear, light gray color. Sections of the wall show hyaline cartilage on one side and a thin outer layer of connective tissue, invested by squamous epithelium and lined by several layers near the cartilage and the large mucous glands of the bottom of the ventricle. The tissues are poorly supplied with vessels.

"The right lung has the usual three lobes, but at the upper part of the hilum on that side and attached by a thin pedicle there is a small extra lobe which reproduces in miniature the shape of the lung, being an oblique and slightly compressed pyramid. There is no trace of a bronchus for this rudiment, but at the lower inner angle an artery enters it which comes from a division of a vessel sent across from the left pulmonary artery; this vessel divides in the apex of the right lung into three main branches just beneath the pleura and from one of these the artery passes to the rudimentary lobe; its vein joins the right innominate vein. The dimensions of the rudiment are 20 m.m. in width, 23 m.m. along the shorter border, and 33 m.m. along the other; the vessels measure 7 m.m. for the artery and 5 m.m. for the vein.

"The ductus Botalli is closed; the aorta appears to give off a bronchial artery proximal to the isthmus. The heart is not included in the specimen and so the relations are somewhat uncertain.

"Sections of the lung show lobular pneumonia.

"The esophagus is apparently normal. The lymph nodes all through the specimen are slightly larger than normal."

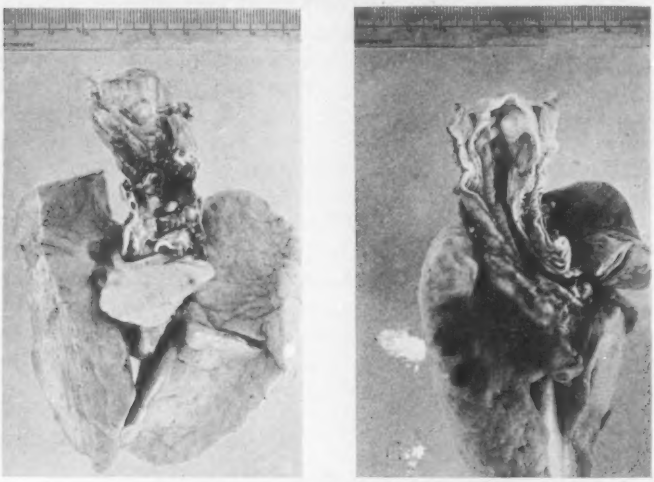
Additional pathological report:

"Section accessory lobe of lung: The section shows numerous tortuous, dilated bronchioles (no cartilage in the walls of any in the section). There also are numerous alveoli about twice the size of the normal for this age. A striking thing is the relatively large number of dilated bronchioles. The walls of bronchioles and alveoli show no appreciable abnormalities."

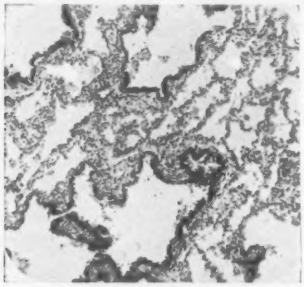
HISTORICAL REVIEW.

New,[1] reporting on cysts of the larynx operated at the Mayo Clinic, states that Gibb operated on a cyst of the vocal cord in 1863, and New cites this as the first case reported in the literature. It is

uncertain whether New means the first published report of operation of a laryngeal cyst or simply the first published report of a cyst of the larynx. But certain it is that the diagnosis and operative treatment of laryngeal cysts are both rare and recent as compared to other established surgical procedures. Ballantyne in his monumental



Laryngeal cyst and lobus azygos of lung. Anterior view.
Laryngeal cyst. Posterior view.



Photomicrograph of section of lobus azygos of lung (mag. about 100x), showing numerous dilated bronchioles and large alveoli.

work, "Antenatal Pathology and Hygiene," doesn't even mention cyst of the larynx.

The cyst, the subject of this monograph, is unusually interesting because of its location in the ventricle. Greene[2] quotes Moure's 117 collected cases of cyst of the larynx (a classical series reported in

1881) as an example of their relative frequency of location: 60 on the epiglottis, 45 on the vocal cords, 8 in the ventricles, 4 on the arytenoids, 3 on the aryepiglottic fold, 1 on the cartilage of Santorini, 2 on the ventricular bands, 4 on locations not given.

Moritz Schmidt in his work on "New Growths of the Upper Air Passages," states that in ten years and in a clinic of 32,997 cases he saw a total of eight cysts, two in men and six in women.

Greene[2] quotes Perseigle, who reviewed all reported cases of tumors of the larynx up to 1905 and found that 40 per cent were cysts.

Chamberlin[3] states that laryngeal cysts comprise 2 to 5 per cent of all benign growths of the larynx.

Ballenger[4] believes that the incidence of laryngeal cyst is greatest in middle adult life.

ETIOLOGY AND PATHOLOGY.

Of the several theories of the etiology of laryngeal cysts some are quite logical, while others obviously fanciful.

Retention cysts develop by the obstruction of a duct of a mucous gland and the retention of secretion. Such cysts, when of short duration, are lined by columnar epithelium, which becomes flattened as the intracystic pressure increases. Retention cysts comprise the majority of all laryngeal cysts. The profusion of mucous glands on the anterior surface of the epiglottis and in the ventricles accounts for the preponderance of retention cysts in these locations, whereas the vocal cords are more often the site of cysts due to the extravasation of blood or lymph.

As to the origin of embryonal or so-called congenital cysts, Schneider believes that they are formed from the pinching off of the ventricle of the larynx. Their branchiogenic origin from the mesodermic tissue of the branchial arches is a theory advanced by Louys. Controversially, the Louys theory is refuted by Von Kostanecke and Mulecki, who claim that during embryonic life the pharyngeal pouch does not come in relation to the trachea or larynx. Glas suggests that the junction of the entoderm and ectoderm at the larynx may be a causative factor, but New objects on the grounds that all epithelial parts belong to the entoderm. Beck proposes the thyroglossal duct tissue as an etiological factor.

Cysts due to the extravasation of lymph or blood are described as being due to a localized effusion of serum between the epithelium and the stroma or an effusion into the stroma. These are frequently located about the vocal cords.

Laryngeal cysts due to echinococcus are rare and the etiology is probably questionable.

Dean and Gregg[5] report a case of an implantation cyst of the larynx in the interarytenoid space following curettage of the larynx for tuberculous laryngitis. Their search of the literature did not reveal a single case of implantation cyst of the larynx, though they state that implanation cysts of the skin and cornea are described.

TREATMENT.

According to New,[1] the best method of treating laryngeal cysts, when possible, is to remove the cyst wall and cauterize the base where the cyst was attached either by the indirect, direct or suspension method, whichever the individual case may indicate. He suggests that a thyrotomy may occasionally be necessary as an avenue of approach. Indeed, this latter method has often been the method of choice of English laryngologists as recorded in their published case reports.

Chamberlin[2] speaks of suspension laryngoscopy as being a boon both to the operator and the patient by facilitating the effort of each. It is certain that with suspension laryngoscopy a large percentage of laryngeal tumor operations are rendered technically easy which otherwise would prove difficult of safe and rapid accomplishment.

LOBUS AZYGOS OF RIGHT LUNG—HISTORICAL REVIEW.

Ballantyne[6] states that malformations of the lungs are usually of the nature of accessory lobes. According to Cunningham[7] "azygos lobe" is a term commonly applied to an accessory pyramidal-shaped lobe occurring on the lower part of the mediastinal surface of the right lung. The first recorded case of lobus azygos was described by Wrisberg in 1778. Stibbe[8] designates an apical lobe fissured by an abnormal "azygos vein," etc. The lobe of the "azygos vein" and those accessory lobes above the root of the lung which are not cut off by the "azygos vein" he calls "upper azygos lobes" in contradistinction to accessory lobes found below the root of the lung, which bear the name of "azygos lobes."

From a review of 25 undoubted cases of lobus azygos, Stibbe constructs the following classification on the basis of the position of the fissure of the azygos vein.

a. More or less horizontal and cutting the outer or lateral surface of the lung at some point between the apex and a position 2 inches below the apex.

b. More nearly vertical and dividing the apex into lateral halves.

c. Vertical and cutting off a small tongue-shaped lobe from the inner or mediastinal surface, the pedicle being attached to the upper margin of the root of the lung.

Thus, a lobe of the azygos vein, Type C, would at once convey the meaning of a small lobe on the mediastinal surface of the lung, attached by a pedicle to the root of the lung and separated by the azygos vein from the upper lobe.

Unfortunately, in removing the specimen herein reported the relation of the pedunculated accessory lobe to the azygos veing was not observed, but in all other respects the specimen fulfills the requirements of Stibbe's Class C.

In analyzing the 25 collected cases, Stibbe found that the type was specified in 19, and of the 19, Type C comprised 20 per cent. Both Cairney[9] and Stibbe believe, however, that a larger series of cases would demonstrate about an equal incidence of all types.

ETIOLOGY.

Stibbe believes that the cause of the abnormality is a failure of the lung bud to clear the venous arch produced by the forward and downward traction on the anterior end of the posterior cardinal vein. He further states that it is not known whether the lung fails to extend so far as usual to the right or whether the vein is already deviated to the right side so that a normally extending lung bud fails to get entirely beyond the vein.

Cleland[10] accounts for the abnormality by supposing that at an early period there occurs a slight adhesion of the lung to the thoracic wall or less probably an undue curvature of the embryo so that the vena azygos as it bent downwards to a position at right angles to its original direction, instead of slipping behind the pleura and lung, dragged down a fold of the former and deeply notched the latter.

Cunningham[7] mentions the constant occurrence of a lobus azygos in certain mammals. Cairney, together with Waterston, Holtby and other observers, affirm that lobus azygos of the right lung is a constant finding in the porpoise. This observation was first reported by Allen in 1882.

CONCLUSION.

This report has dealt with the presence of:

1. A congenital laryngeal cyst of the ventricle occurring in an infant, who died at the age of 6 weeks, of obstruction to respiration, together with a bronchopneumonia.

2. A lobus azygos of the right lung.

The writer gratefully acknowledges the assistance of Dr. B. S. Kline, Pathologist of Mt. Sinai Hospital, and Dr. A. E. Thayer, Pathologist of St. Luke's Hospital, the former for the excellent

photographs and photomicrographs, and the latter for the pathological report.

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10208 Euclid Avenue.

SUPPURATIVE LABYRINTHITIS. SEROUS MENINGITIS. RECOVERY.*

DR. RICHARD TRAVIS ATKINS, New York City.

M. H., female, age 20 years. Admitted to the New York Eye and Ear Infirmary, August 2, 1926, and discharged Sept. 18, 1926.

Previous History: Meningitis at age 5 months. Measles at age 3 years, complicated by bilateral otitis media and followed by impaired hearing. Right simple mastoidectomy at age 17 years, with good recovery. Intermittent discharge from the left ear for the past year.

Present History: Began four days prior to admission to the hospital with pain in the left ear, followed by fever and an occasional attack of dizziness. On the day before admission the patient became nauseated and vomited.

Examination: The temperature on admission was 103.2° F., the pulse rate 120, and the respirations 20.

There was a profuse, foul, purulent discharge from the left ear. The middle ear was filled with necrotic granulations. Smear examination of the pus showed a mixed infection.

The leucocyte count was 19,200, 88 per cent of which were polymorphonuclears.

The left labyrinth was active on functional examination, and there was hearing for the whispered voice at the meatus. There was no spontaneous nystagmus. The fistula test was not made.

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X-ray examination showed a sclerotic mastoid process on the left side with a far forward sinus. The upper part of the petrous base showed an excavation.

Operation: An immediate radical mastoidectomy was performed by one of the members of the staff. The mastoid process was sclerotic. There was a large cholesteatoma in the antrum which extended into the middle ear. The dura was found exposed above the antrum, and there was a fistula present in the external semicircular canal.

The usual radical procedure was followed, and the fistula left undisturbed.

Course: Following the operation the patient complained of dizziness, headache, nausea and vomiting. The temperature remained high.

On the second day after the operation there was some rigidity of the neck and a slight rotary nystagmus to both sides. The spinal fluid was under considerable pressure, slightly cloudy, and contained 180 cells to the c.m.m. Cultivation was negative. The Wassermann reaction was negative. Hearing in the affected ear was still present and the tuning fork lateralized to the diseased side.

Two days later (four days after operation), the spinal fluid contained 280 cells. The symptoms continued as before with the exception of the nystagmus, which was now toward the sound side only. Hearing was still present. The eye grounds showed a slight blurring of the discs, but no elevation.

The spinal fluid was withdrawn regularly every other day, and the cell count gradually increased to 58,600. Cultivation of the spinal fluid continued negative. At this time (two weeks after the operation), the hearing disappeared, and the hot caloric (112° F.) failed to influence the nystagmus, which was still present, but not as active.

During this period the patient was nauseated and vomited frequently. She complained of severe headache and backache. The temperature varied from 98.2° F. to 104° F.

Because of the nonreactibility of the acoustic and vestibular labyrinths a second operation was performed, which consisted of a labyrinthotomy of the Hinsberg type. Liquid pus was found in the vestibule.

Gradually the symptoms abated and ten days after the operation the cell count had dropped to 12,200. No further spinal punctures were made because the patient refused to co-operate.

The patient was discharged from the hospital on Sept. 18, one month and a half after admission, in fair condition. The wound was still granulating and was subsequently dressed by an otologist in her home town. A complete recovery has been reported.

4 West 53rd Street.

a. MASTOIDITIS WITH GRADENIGO SYNDROME BEFORE OPERATION. b. MASTOIDITIS WITH GRADENIGO SYNDOME FOLLOWING OPERATION.*

DR. J. D. WHITHAM, New York City.

Since 1904, when Gradenigo first ascribed the symptom-triad which will always bear his name, over three hundred cases have been described in the world's literature. A recent thesis by W. H. Sears describes the condition in a most exhaustive way. Prior to this the late Dr. Perkins and Dr. John Wheeler, of New York, made valuable contributions to the subject. It is quite a rare condition but frequent enough to warrant inclusion in all textbooks of otology and ophthalmology. An isolated paralysis of the abducens nerve means a lesion of the peripheral portion of the nerve, for a lesion of the intracerebral portion would always cause a conjugate palsy. Some fifty causes could be given for an isolated abducens paralysis, but when it is associated with a discharging ear and deep temporal pain the diagnosis of Gradenigo's syndrome is easy. In Kollner's study of 250 cases of paralysis of the externus, only 2 per cent were associated with otitis, which indicates the rarity of the syndrome under discussion. The majority of cases are probably due to an osteitis at the apex of the petrous bone with an adjacent pachymeningitis; some are associated with extradural abscess, some with phlebitis of the venous channels of the carotid canal via the peritubal cells. The streptococcus mucosus is very frequently the offender. The mortality is about 18 per cent.

Case a: Mastoiditis with Gradenigo syndrome before operation. Mrs. D. A., age 23 years, April 1, 1923, patient referred by Dr. Frank Keil.

Previous Medical History: Six weeks ago was in bed for five days with grippe and cold in the head. At that time she had a sudden severe pain in both ears and both drums were incised. Two weeks later the right drum was again incised, for the pain had returned. During that interval the left ear ceased discharging. For the past two weeks she has had continuous discharge from the right ear and very severe pain in the right temporal and right frontal region. She told me that she had had fever until ten days ago, but since then had

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not felt feverish. She stated that small doses of aspirin easily controlled the temporal pain.

Examination, April 1, 1922: Healthy looking young woman. Temperature 98.8.° Pulse 82. Pupils, ocular movements and fundi negative. Slight tenderness and pain on percussion over right temporal and parietal region. No tenderness over mastoid bones, but there was a suggestion of a loss of mobility of the soft tissues over the right mastoid. There was a profuse whitish aural discharge from the right ear, with a thickened edematous drum and a small perforation in the posterior inferior quadrant. The canal was not narrowed. The watch could be heard at 3 inches and the whispered voice at 4 feet. The left ear was normal.

On April 3, two days later, the patient first noted a diplopia and on examination on April 4 a well marked paralysis of the right external rectus was present. She told me the temporal pain was more severe and extended into the right upper jaw and frontal region, being most severe, however, in the temple. Eye examination was negative except for the paralysis noted above. The local signs in the ear and mastoid showed no change. The X-ray showed a cloudy right mastoid of pneumatic type, with a suggestion of softening at the knee of the sinus. Pulse and temperature were normal. General physical, urinary and neurological examinations were negative.

On April 5, a right simple mastoidectomy was done. Free pus was found on removal of the cortex. The entire mastoid and petrous was extremely cellular, the spaces extending far forward into the zygoma and very deeply into the petrous below and mesial to the horizontal canal. The bone showed some softening in the region of the antrum and sinus plate. There were no exposures of sinus or dura.

On April 12, the function of the external rectus had returned and diplopia was seldom noted by the patient. The temporal pain had ceased and on April 16 the patient left the hospital. The wound healed rapidly and when seen one year later she had had no further trouble.

Case b: Mastoiditis with Gradenigo syndrome following operation. In the second case the externus paralysis did not begin until four weeks after I did a simple mastoidectomy. This patient, Richard H., age 34 years, was admitted to the service of Dr. Dench at the New York Eye and Ear Infirmary on June 22, 1926. He stated then that five weeks previously he had had a cold in the head with pain in the right ear. The right drum was incised three days later. The discharge ceased after a day or two, but returned two weeks later and has been profuse ever since. For the past week he has had postauricular pain and tenderness. Examination of the left ear was nega-

tive. The right ear showed a posterior slit-like perforation with profuse ropy discharge and bright red drum. The canal roof did not sag. Postaural tenderness was present. The tonsils were large and cryptic. General physical examination and urine examination were negative. Smears and culture from the canal showed the pneumococcus mucosa. X-ray report on day after admission was as follows:

"Left is a well developed, fairly clear pneumatic mastoid with rather thick septa. Sinus probably forward. *Right* is a similar bone, with more zygomatic cells. Sinus apparently forward. It is exceedingly cloudy throughout without definite indications of softening."

On admission the temperature was 101.9,° pulse 84, and respiration 20. On June 23, a right simple mastoidectomy was performed. Free pus was found in the cells and antrum. A large perisinus abscess was present, which resulted in the exposure of about 1 inch of sinus. The tip and zygoma were carefully cleaned out. The dura was not exposed. The patient recovered quickly from the operation and left the hospital on July 6 in excellent condition, with a dry canal and with the posterior wound clean and apparently almost healed. He was dressed in the dispensary and the discharge from the wound increased in amount, but the canal remained dry until July 26, when he had a return of pain in the right ear and abundant discharge. The next day he felt a very severe pain over the right eye, and on July 28 he felt dizzy and had double vision. He spoke of a thumping noise in his ear. The pain continued with increasing severity and was steady, being in the right forehead, face and right temporal region. He appeared quite sick, his face being drawn and thin and pale. The mastoid wound was reddened, but no pus could be expressed. The canal was dry. There was a slight sagging of the posterior canal and a lustreless drum with posterior perforation. A slight induration and tender spot was noted about 2 inches below the wound. An apparently complete paralysis of the right external rectus was noted. Percussion on the right temple and right forehead caused him to wince. Physical and neurological examination was carefully made but revealed no further abnormality.

Eye examination was as follows: "O. D. Disc-margins somewhat indistinct, more marked to nasal side. Slight enlargement of lower branch of retinal vein. Appears to be a very early stage of papilloedema. O. S. High myopia with chloroidal stretching temporal side. Unable to get a good view of disc as patient could not keep eyes in one position. Paralysis of external rectus right eye."

Functional examination of ears showed, right ear: watch, 6 inches; low whisper, 2 feet. Increased bone conduction and lateralization of

Weber to the affected ear. Left ear was functionally practically normal. Caloric reactions were normal for both ears.

X-ray, July 30, 1926: "Right mastoid shows a large cavity indicating that the cells have been rather thoroughly cleaned out in the mastoid region. There is an area about 0.5 m.m. broad over where the lower part of the sinus should be, suggesting removal at that point. There is also some indication of a few remaining zygomatous cells above the antrum and over the region of the canal."

X-ray of the Nasal Sinuses: "Frontals are moderate size, and not particularly clear. Ethmoids are broad and clear. Considerable thickening in the antra, especially on the right side."

July 30: "Spinal fluid clear. Globulin negative. Fehlings reduced. No cells. Cultivation negative."

Blood count, July 30: Hgb., 90 per cent; red cells, 4,500,000; white, 8,000; small, 31; large, 6; trans., 2; poly., 5; eosin, 0.

Urine examination negative. Temperature on second admission, 100⅖,° pulse 102, respiration 22. On July 30, 1926, a secondary simple mastoidectomy with exploration of the middle fossa was done. The old mastoid incision was reopened and extended forward over the zygoma. Exposed sinus appeared healthy. Considerable necrosis was found in the region of the antrum. Zygoma and region of posterior semicircular canal were cleaned out as far as possible. The dura was freely exposed in the middle fossa and the opening extended as far forward as possible on the anterior surface of the petrous portion of the temporal bone by removal of squamous plate above the zygoma. Probing in the direction of the Gasserian ganglion revealed no pus. On cleaning out cells deep down in the direction of the jugular bulb several distinct twitchings of face were noticed. One strip iodoform gauze was used for packing. Closure by means of three SWG sutures and Michel clips."

The impression was gained that the entire petrous bone was pneumatic. He showed no improvement following the operation until after one week, when his dizziness ceased, the pain almost stopped and his temperature became normal.

On August 9, 1926, he was discharged from the hospital. At that date he showed a slight return of function in the external rectus muscle. The canal was slightly moist and the postaural wound was discharging. Following his departure from the hospital the patient was dressed in the dispensary. There was a gradual improvement in the ocular paralysis and in the condition of the wound until Sept. 25, 1926, when he left the city, apparently almost well.

TWO CASES OF CAVERNOUS SINUS THOMBOSIS, ASSOCIATED WITH (1) ACUTE PURULENT OTITIS MEDIA; (2) MASTOIDITIS.*

DR. WESLEY C. BOWERS, New York City.

Case 1: B. H., age 27 years, was admitted to Bellevue Hospital, Oct. 30, 1926. Pain started in the left ear the morning before, at which time the patient came to the O. P. D. Examination showed the left drum red and bulging; it was also noticed that his eyelids were puffy, although no significance was attached to this at the time. He was sent up for myringotomy, but went home instead.

The following morning he was admitted to the hospital. The drum had ruptured spontaneously during the night. He had a temperature of 102,° pulse 110, respiration 22. He complained of feeling chilly and of pain over his left frontal region. His left eye was edematous and there was marked proptosis; the right eye was also protruding but less markedly until the evening. Examination of the left ear showed a profuse discharge in the canal. A spinal tap gave increased pressure, fluid uniformly cloudy; cells, 18,000 per c.m.; direct smear showed a majority of polymorphonuclears, with a few extra cellular diplococci; culture was negative. Examination of the eyes later in the day showed small pupils, which reacted to light; fundi showed dilated veins, O D > O S. There was no edema of the nerve head. There was chemosis, double ophthalmophelgia and proptosis. The patient was very restless during the day, complaining of frontal headache extending back to the occiput. Toward evening he was restrained and given Magendie. That night he became unconscious and died at 12:45 the next day.

This case was one in which the ear and cavernous sinus conditions were concomitant. The first symptom was pain in the left ear; forty-eight hours later the patient died with all the symptoms of cavernous sinus thrombosis and meningitis.

Case 2: F. K., age 33 years, was admitted to St. Luke's Hospital on Oct. 16, 1926. He gave a history of intermittent discharge from the ear for four years. Eight days previous to admission, he had a chill and pain in the left ear. This condition cleared up during the day, recurring the following day. The third and fourth days, the

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patient went to work but had some earache the fourth day. On the fifth day, he began having pains all over the head, associated with vertigo, and a temperature of 102° to 104.°

Physical examination revealed a marked spontaneous nystagmus to the right. Fundi, lids and conjunctiva were negative. The left ear had a large polyp in the meatus, hanging from the superior wall; the drum could not be seen. There was profuse, purulent discharge. There was tenderness on pressure over the mastoid about an inch posterior to the auricle, on a level with the canal. The spoken voice was heard at a distance of 4 feet. The labyrinth was active. Spinal fluid showed 25 cells; Butyric negative; culture negative. The blood count was 7,350; polys. 76 per cent; lymphocytes 24 per cent; blood culture was negative. Culture from the aural discharge showed Gram negative bacilli.

Operation, performed the following day by Dr. Manheim, revealed the following pathological findings: Upon removal of the cortex, there was an escape of pus and gas under pressure; the odor was that of colon bacilli, but culture from the wound showed no growth. The sinus was completely collapsed, contained no blood clot and the outer wall was necrotic. Upon insertion of the probe toward the torcular, free bleeding was obtained; likewise from the jugular bulb. The diseased cortex was removed anteriorly and posteriorly from the sinus, exposing the dura for 2 c.m. in each direction. A few granulations were observed upon the exposed dura in the middle fossa. Operation had to be discontinued at this point because of the condition of the patient. The jugular vein was not ligated.

Two days later, the spinal fluid showed 450 cells; 98 per cent polys. There was ophthalmoplegia of the left eye, the veins of the fundus were markedly congested. There was Kernig, stiff-neck, restlessness and headache. The blood count was 25,700, 95 per cent polys.; blood culture still negative. The temperature throughout ranged from 101° to 108.° Death occurred the third day after operation, with all the symptoms of meningitis and cavernous sinus thrombosis.

This was a case in which the involvement of the cavernous sinus probably took place through the petrosal. I have seen two other cases of cavernous sinus thrombosis, both following improper treatment of a nasal furuncle. All four cases died within 48 hours after the first appearance of eye symptoms.

Cavernous sinus thrombosis was first recognized in 1821. There have been reported in the neighborhood of 300 cases from all causes, of which the most common was some involvement of the face, eye or

nose, especially following incision of nasal furuncles. A possible anatomical reason for this association of facial conditions and the cavernous sinus is the fact that the veins leading from the face to this sinus are valveless. As a complication of mastoiditis, it is a rare condition. This is clearly evidenced by a careful search of the literature which has revealed only twenty-six cases secondary to mastoiditis. About fifty-two autopsies have been reported in which cavernous sinus thrombosis was associated with an aural condition. This leaves twenty-six cases which originated from ear conditions other than mastoiditis.

In the cases associated with mastoid, the onset has been from twelve hours to twenty-five days, after operation. The average has been about two days. Duration of the illness has been from two days to several weeks after its recognition, the average being about three days. In all cases, the usual symptoms are ophthalmoplegia, proptosis, chemosis, tortuosity of the veins of the fundus, with possibly retinal edema and hemorrhage. At the same time, Jansen has proven by autopsy that purulent thrombosis of the cavernous sinus is possible with the absence of any change in the fundus, or any impairment of the visual acuteness, or the function of the ocular muscles.

The organism in most cases has been streptococcus, found only at autopsy from the clot. The absence of a positive blood culture in these cases is surprising. In the few instances in which the blood culture was reported, as also in the two I am citing, all blood cultures were negative. The blood count has shown a lymphocytosis of from 16,000 to 40,000 cells, with polymorphonuclears between 76 and 90 per cent.

The commonest route taken by the infection seems to have been through the superior and inferior petrosal sinuses, although one case is given in which at autopsy, no involvement of the lateral sinuses was found, while the superior petrosal contained a thrombus. Another case, at autopsy, revealed no involvement of the lateral sinus. The incidence of cavernous sinus occurring in conjunction with lateral sinus thrombosis is 50 per cent.

The prognosis is almost always fatal. Three or four cases have been reported of spontaneous recovery following ligation of the jugular and drainage of the lateral sinus. There is one case cited of recovery following drainage of the cavernous sinus; drainage was effected posteriorly through the petrous bone, but this was followed by permanent facial paralysis and ophthalmoplegia. The approach has been through the Hartley-Krause gasserian ganglion route,

through the orbit, through the sphenoidal sinus and through the petrous portion of the temporal bone.

Conclusions: Cavernous sinus thrombosis as a complication of mastoiditis is very rare. It may be present but not suspected in many cases, as evidenced by the number of autopsies which have revealed it when it had not been suspected.

It may be secondary to aural conditions other than mastoiditis.

It is frequently present without involvement of the lateral sinuses.

Positive blood cultures are seldom found.

Diagnosis may be impossible, as the thrombosis can exist without any eye symptoms.

Inasmuch as operative drainage of the cavernous sinus has met with failure in all cases but one, it would seem advisable to try and deal with cavernous sinus thrombosis in some other way or by some different operative procedure. Since it occurs so frequently independently of lateral sinus thrombosis, it would seem that the only rational operative procedure would be either to drain the cavernous sinus, or to enable nature to take care of it by putting it at rest as much as possible. This could be accomplished to some extent by ligation of the internal carotid.

Among all the cases of cavernous sinus thrombosis from all causes, only one has recovered as a result of operation on the sinus; about fifteen have recovered spontaneously. This would seem to indicate that the conservative treatment was best.

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17 East 61st Street.

TUBERCULOSIS MASTOIDITIS.

Dr. T. R. Chambers, Hamilton, Bermuda.

A girl, age 15 years, was referred to me by Dr. W. E. Tucker. Her history and temperature chart tell the story. She first came to me on March 30, 1926, complaining of pain in her left ear, deafness and fever.

History of Present Illness: Eight days ago she began to have pain in her left ear, without any previous cold or cough. The pain was intermittent, with noises and throbbing in ear at times. She had been in bed since the pain started. Had only slight headache and no digestive disturbances until today, when she vomited. No pain behind the ear.

Past Illnesses: Previous trouble in same ear several years ago, while living in Portugal; suppuration of a left cervical lymph gland several years ago. She has lived in Bermuda for three years.

Family History: Father, mother, brothers and a sister living, but none of them looks robust. No deaths in family. The family works on a farm.

Physical examination shows a small Portugese girl, pale, but not badly developed except that she is small.

Scalp, eyes and nose negative. Ears: She cannot hear a watch tick by her left ear. Left posterior wall of auditory canal is bulging and when incised released seropurulent fluid. Mastoid not tender. Right ear slightly deaf. One carious tooth; tonsils and pharynx negative; small cervical lymph glands with a scar at base; thorax symmetrical; lungs resonant and clear throughout; heart negative; abdomen negative; left epitroclear nodes are palpable; knee jerks active; no edema of extremities; urine amber color, acid, no albumin or glucose, few W. B. C., very few R. B. C., many urates. Blood count: W. B. C. 7,000. P. M. N. 87 per cent. S. L. 8 per cent. L. L. 5 per cent. March 31: W. B. C. 6,800. P. M. N. 83 per cent. S. L. 11 per cent. L. L. 5 per cent. Bas. 1 per cent. Stool: Ova of trichiuris trichiura found. Blood: Formalin test for syphilis negative. April 2: W. B. C. 6,100. P. M. N. 77 per cent. E. 1 per cent. S. L. 21 per cent. L. L. 1 per cent.

Her ear continued to discharge, but she seemed quite comfortable until April 10, when her fever increased without any change in the signs about the ear, which discharged as usual. She had been up and about the ward, but now was put back to bed again.

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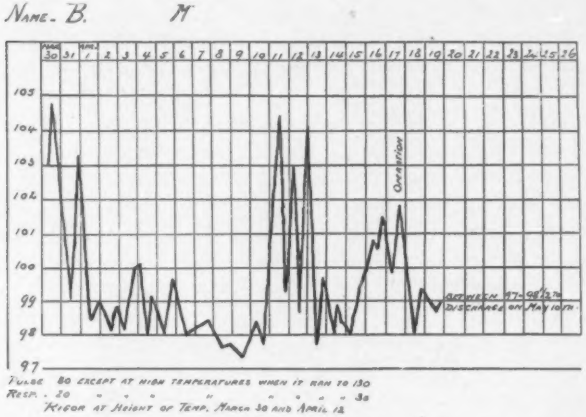
On April 11, patient vomited several times; ear discharging; no mastoid tenderness. Blood count: Hgb. 60 per cent T. W. B. C. 16,000. P. M. N. 87 per cent. L. L. 8 per cent. S. L. 5 per cent. April 12: Chill; ear discharging; no mastoid tenderness; throat negative. Blood count: W. B. C. 12,200. P. M. N. 86 per cent. S. L. 8 per cent. L. L. 3 per cent. Tr. 1 per cent. B. 2 per cent. Three days of practically normal temperature followed. April 16: Fever. April 17: Blood count: Hgb. 65 per cent. T. W. B. C. 8,200. P. M. N. 70 per cent. S. L. 20 per cent. L. L. 8 per cent. B. 2 per cent.

This operation consisted simply in opening a 2 m.m. cavity filled with pus just before reaching the antrum; cavity was curretted and packed with iodoform gauze. Wound healed in ten days. The mastoid was very hard bone. The blood, when cultured on agar and


Name. B. M
Operation
Discharged on May 10th
Pulse 80 except at high temperatures when it ran to 130
Resp. 20 " " " " " " " 30
Kidgor at Height of Temp. March 30 and April 12

blood agar failed to produce any growth. Tubercle bacilli alone were found, according to Dr. Henry Wilkinson. April 20: Smear of pus from mastoid wound showed G+cocci and bacilli and G—bacilli. Carbo-fuchsin stain showed a good many acid-fast bacilli. April 23: Blood count: Hgb. 75 per cent. W. B. C. 9,800. P. M. N. 65 per cent. E. 1 per cent. S. L. 20 per cent. L. L. 5 per cent. May 3: Mastoid wound has healed; ear still discharging. Smear from ear shows G+cocci and G+large bacilli (too large for tubercle bacilli), small G—bacilli, the latter predominating; no pus cells. Weight 85 pounds. May 10: Discharged. General condition very much improved; right ear seemed to be normal and hearing in left ear very much improved.

AN IMPROVED HYPOPHYSCOPE.*

Dr. W. Harry Barnes, Philadelphia.

The work of Cushing has proven that the trans-sphenoidal approach to the pituitary gland is the route of choice. Cushing makes an incision below the upper lip and from there does a submucous

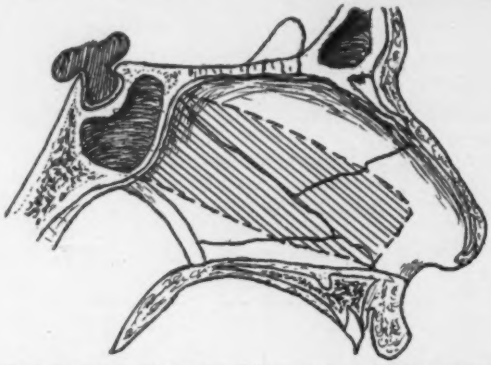


Fig. 1. Shaded area is the part of the nasal septal framework removed by Segura in his transphenoidal approach to the pituitary gland.

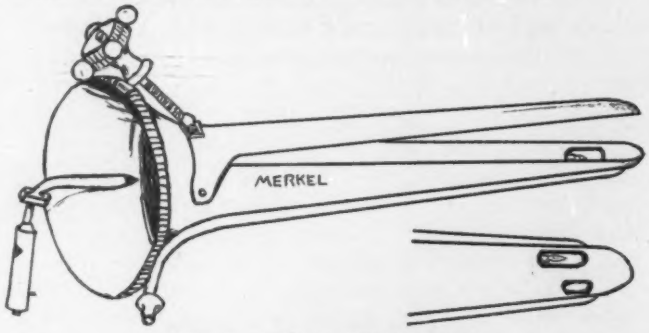


Fig. 2. Author's Hypophyscope.

resection of the nasal septum to reach the sphenoid sinuses. Hirsch resects the septum from the usual intranasal incision for a submucous resection. Segura has improved the Hirsch operation, and the new instrument is an improvement of the one used by Segura.

*Demonstrated at the meeting of the Philadelphia Laryngological Society, May, 1926.

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The hypophyscope is a bell-mouthed bivalve speculum. The fixed blade has attached to the outer surface two canals which open on the inner surface of the end of the blade. One is for the light carrier and the other is for suction and is continued so as to fit the suction tube of the suction apparatus.

The distal lighting and suction canals are principles borrowed from the Chevalier Jackson endoscopic tubes and are made on the same general principle. Light is very necessary in pituitary operations, and, as they are very bloody, continuous suction saves much time.

The hypophyscope is made in two sizes: large (Fig. 2) for the Cushing incision, and small for the Segura incision. Mr. Edward Merkel has aided me in overcoming many of the practical difficulties in the construction.

1315 North 15th Street.

A NEW ANTROTOMY KNIFE AND A NEW ANTRUM CANNULA.

DR. ELLIS M. SCHWARTZ, New York City.

1. The knife is double bladed, has a trocar point and a heavy handle for a firm grip.

Some advantages derived from the use of this instrument: *A.* Easy entrance into the antrum. *B.* No second instrument needed for enlarging the opening. *C.* By being pulled forward, the knife makes a clean double incision into the nasal wall of the antrum, removing a strip of bone .3 c.m. wide, and as long as is desirable. *D.* On removal of the knife, the strip of bone is either caught on a small hook protruding from the trocar point and comes out with the knife, or may remain hanging in the inferior meatus. This can then readily be removed with a pair of grasping forceps. *E.* No shock to the patient, as no biting or rasping is done.

2. The cannula is a modification of the Eicken's, a commonly used cannula which has its opening at the tip of the instrument. In my

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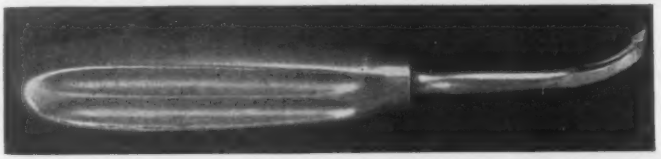
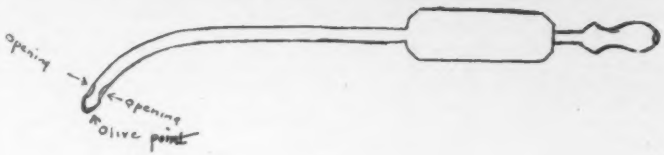
instrument, as illustrated, I have made the following changes: I have made tip olive-pointed, like a probe; instead of one opening there are two, one placed opposite the other.

Advantages: A. This tip enables the instrument to slip into the antrum, through any size opening—like a probe.





B. The position of the openings enables us to give the antrum a better flushing, since the streams are in two directions.

C. In sinuses, where there are polyps, when the old cannula is used there often is interference with the flow of the solution into antrum. The tip of this instrument displaces the polyps and they cannot interfere with the flow, as the openings do not come in contact with them.

These instruments are made by Pfau's American Instrument Company, 2 East 23rd street, New York City.

308 East 15th Street.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, Dec. 22, 1926.

Left Recurrent Laryngeal Paralysis with a Questionable Early Carcinoma of the Larynx. Dr. George D. Wolf.

I feel that I owe some apology to the members and guests of this Section for presenting this rather not uncommon case, but I have done so for two reasons: First, because we are constantly urged by the medical as well as the lay press to make an early diagnosis of carcinoma, and as laryngologists we feel that we should do our share; second, can one make an early diagnosis of carcinoma in an approachable region without the support of the pathologist? I have had some disagreeable experiences in this respect.

About two years ago, I presented here a man, age 45 years, who had a similar lesion as the one I am about to present this evening, but who clinically did not look like a case of carcinoma, still two of the pathologists in one of the hospitals I am connected with made a diagnosis of early carcinoma on a specimen removed from that larynx. Dr. Kramer, who was present at the meeting, asked me to let him show my slide to the late Dr. Mandelbaum, who made a diagnosis of chronic inflammatory tissue. I saw this man about a year later and his condition was about stationary.

A short time after that I saw a man, age about 50 years, on whom I made a diagnosis of intrinsic carcinoma of the larynx. In order to verify the diagnosis, I removed a piece and sent same to a competent pathologist, who diagnosed same as being a papulofibroma. In view of his age, I urged my patient to have the growth removed, lest it become malignant. He left me, and as Dr. Jones told me subsequently, applied to his clinic and the pathologist there made a diagnosis of carcinoma both before and during the operation.

The patient I am presenting here tonight is from Harlem Hospital, service of Dr. Hubby, female, age 27 years, single, registered nurse by occupation, somewhat self-centered and wants a lot of medical attention, and as she is personally acquainted with many physicians she gets it. She denies venereal history; she says that numerous Wassermanns done on her were negative. She gives a history of having had pneumonia three years ago, which was followed by double mitral lesion. Two years ago she developed an embolus of the brain, followed by hemophlegia, which cleared up subsequently. About two years ago she was in another hospital, where she was treated for decompensation of her heart and while there complained of pain in her larynx. She was examined by a laryngologist and was told that she had a left recurrent laryngeal paralysis. This fall she had pneumonia and was confined in the Harlem Hospital for over eight weeks. While at the Harlem Hospital she complained a good deal of hoarseness and pain in the larynx. Examination of her larynx revealed a complete left recurrent paralysis and a wartlike growth in the interarytenoid space. Six weeks ago, she had a specimen removed from her larynx in one of our large hospitals where malignancy is treated. The pathologist there, one of the most prominent men in the city, said that we are probably dealing here with a precarcinomatous stage and advised radium treatment. She has already received one treatment about a week ago. Three weeks ago, I removed a specimen from the interarytenoid region and the local pathologist thought that it is probably a case of early carcinoma. In order to make this diagnosis certain, he showed the slide to Dr. Simmers, who pronounced same as being a papulofibroma. The patient insists on having her larynx removed now rather than wait till the carcinoma will develop. Personally, I am opposed to this procedure because of her age and because the lesion is located in a place where it is claimed carcinoma never occurs. I believe her paralysis is due to pressure by a large heart—of cardiac origin—and I'm inclined to watch her for further developments. But the outstanding

fact is that we couldn't get two prominent pathologists to make the same diagnosis.

DISCUSSION.

DR. FORBES: I would suggest to Dr. Wolf that he watch the case for another month or two and present her again. Clinically, I cannot believe that we are dealing with a malignant growth of the larynx. The history of trauma which she may have had may account for the slight ulceration. I hope Dr. Wolf will continue to work up the case and present her again a little later. I certainly would not advise nor suggest the removal of the larynx. The question of a further biopsy best be kept in mind.

DR. WOLF: There was no infiltration around the lesion before the radium was applied. Whenever a question comes up as to the difference of opinion given by the pathologists the remarks, like this evening, are usually made that maybe one failed to remove the specimen from the malignant region. I would like somebody to explain to me how the following discrepancy can occur:

A young man, age 17 years, who had three nasal operations before I saw him, the last one having been performed only three weeks ago, presented himself with a huge polyp filling up the entire right nasal vestibule. As I never before saw polyps of that size to recur so rapidly, I somewhat hesitated to go ahead with him and I removed a specimen of the growth for examination and also blood for Wassermann test, which was sent to the health department laboratory. The report given on this specimen was 2 plus. As there was no venereal history or anything else to suggest the boy being syphilitic, I urged the parents to have another specimen taken, half of which was sent to the health department laboratory and the other half to a private laboratory. The former sent a report of the specimen being 3 plus, and the latter sent a report of the specimen being negative. The situation being more complicated at this stage, I had a third specimen of blood removed and against sent one-half to the health department laboratory under an assumed name, and the other half to another serologist in whose work I have explicit faith. This time the health department laboratory report was 4 plus and the report of the outside serologist was negative. I then communicated with both the health department laboratory and the private pathologist, telling them of the unusual discrepancy. They both tested the same specimen over again and insisted that they were correct.

A short time after that I saw a middle aged lady who had an obscure lesion on the septum which appeared to me to be either a gumma or malignant growth. In view of numerous scars on her legs, also because of her history of arthritis, I thought that the lesion is probably syphilitic. I removed blood for Wassermann and because of the above described experiences I sent part of it to the health department laboratory and the rest to a serologist. The latter gave a report of 4 plus, while the former gave a report of negative. The lesion cleared up promptly on specific treatment. Here, no argument can be given that the specimen removed was not from the right place. Apparently, our laboratory techniques are not perfect. Our pathologists have their own individual views as to what constitutes malignancy and what not. Like clinicians, they are not infallible and therefore the checking up is most essential. Not getting any decided aid from the pathologists, we probably will not make very much progress towards irradicating malignancy in its incipiency.

Returning back to our own patient presented here this evening, while I don't think the lesion syphilitic, I'll put her on specific treatment for a short time on general principles, and will present her here in the near future for a second time.

I thank the gentlemen for participating in the discussion.

Malignancy of the Larynx and Esophagus Treated by Radium Emanation.
Dr. Frank R. Herriman.
(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. T. J. HARRIS: I have followed the reading of this paper with a great deal of interest. Dr. Herriman is certainly to be commended for the industry and enthusiasm with which he has attacked from a new angle a problem that men like Dr. Robinson, Dr. Willis, Dr. Imperatori and Dr. Forbes have been

working on for many years. I listened with a great deal of interest to his conclusions. We must remember that anything that is said here in this Section is going to be heard all over the world. If a statement is made which is going to be misunderstood as a claim for a cure for cancer by radium by Dr. Herriman, it would be most unfortunate. We all understand that. The use of radium in the larynx has received a great deal of consideration in the last ten years. Recently I have been going over the statistics of the work that Dr. Willis did at the Post-Graduate, and Dr. Robinson and Dr. Imperatori at the Manhattan, which stands out as the best that has been done. No pains have been spared to do the work in the best possible manner, nor in keeping the records.

Imperatori reported at the Montreal meeting of the American Laryngological Society last June the results in thirty cases of CA treated by radium at the Manhattan Eye, Ear and Throat Hospital and we were able to obtain records more or less complete of thirty cases thus treated at the Post-Graduate Hospital. Of the sixty cases from the two hospitals, only two were classified as cured for five years or more, one by Dr. Imperatori and one by Dr. Forbes.

The one by Dr. Forbes is known to most Post-Graduate men. She told me not long ago, that if she had to go through the same thing again she would never have the radium applied. I believe that Dr. Forbes feels that the radium had a very good effect. Dr. Imperatori stated that it was doubtful if the radium was the real cause for the cure in his case. So, I should say, based on the work of the last ten years, there have been no statistics to show that radium is a cure for cancer.

Radium in operable cases should not be substituted for operation. I don't understand that Dr. Herriman has made that claim, but I want to strengthen the statement that it is not to be recommended for a cure in operable cases.

It is a question whether it is warrantable to use it as a palliative measure. Up to tonight, the results have been disappointing, in that the immediate results by burns and otherwise have been disastrous, though its use has been most scientific, as the applications made by Dr. Willis, Dr. Robinson and Dr. Imperatori have been carefully guarded against any reaction. It seems to be the peculiar nature of radium itself in attacking tissue, not merely to attack the cancer cells but the healthy cells, and because of the greater strength of the cancer cells, they survive while the others go down.

So far as Dr. Herriman's immediate results are concerned, I think they are encouraging, but to draw any conclusions other than that, he has not had these reactions which too often has been the case with the older methods, is not warrantive. I could not see in any of the larynx cases presented tonight what could be called a cure. In every case presented I should say the diseases was still evident. I don't think there was sufficient encouragement to lead us to expect two years from now to see any of these cases cured. Dr. Robinson could tell you that in many cases he has had no greater reaction than seen in these cases tonight. I do feel that we have a new and admirable method here, but I want to emphasize the point that we should not get from the work the idea that there is a cure, or that the possibility of cure is so great that we can tell our patients or tell others that there is a justification for the use of radium where operation is indicated. Second, that so far as its value as a palliative agent is concerned, it is encouraging, but still too early to say anything but that violent reactions have not been present.

DR. JOSEPH MUIR: Radium needles are about 2 c.m. long and they vary in radium content from 5 to 25 m.g. Due to the large bulk of radium compared with radium emanation, the radium needles were large. To keep the outside diameter of the needle within practical limits, only .2 m.m. metal screenage was used. This metal screen was generally monel metal or steel; the .2 m.m. thickness being equivalent, in filtration power, to only .05 m.m. of platinum. Large intratumoral dosages were given, allowing 25 m.g. needles to remain in place for twenty-four hours—delivering 600 m.c. hours per needle. These had to be placed close together to obtain proper intensity of Gamma radiation within the tumor mass. The result was that due to the improper filtration the intensity of beta radiation was very high and too much causticity resulted. Burning was inevitable and if the needles were placed too close to cartilage and bone in treating laryngeal carcinoma, perichondritis and periostitis was

caused. The caustic beta radiation limited the gamma radiation dosage which could be delivered with an improperly filtered needle, with the result that definite, adequate dosages were never delivered. The biggest dose at one time was that delivered to annular lesions of the esophagus. I have in mind cases treated by intraesophageal applicators properly immobilized and at the most the surface radiation received by fractional dosage was 1,500 m.c. hours delivered over thirty days in several applications. During this time the lesion received no adequate dosage except, possibly, the esophageal lumen.

I have before me a report of Dr. Strauss, of Tenon Hospital, in which a large esophageal mass with complete stenosis was treated for thirty days, both by applicators introduced within the lumen of the esophagus and by surface radiation on either side of the spine, crossfired by an applicator on the sternum. The total dosage given was 80,000 m.c. hours. This dosage must, however, be considered as surface radiation.

I have also in mind cases of leukemia treated by Dr. Burnham which received 160,000 m.c. hours surface radiation. I am here speaking of largess of dose to compare with the puny 1,500 m.c. hours radiation we deliver with needles.

Strauss' case up to this moment has recovered, with the mass regressed, and the patient comfortable.

We must bear in mind that we are dealing with a malignancy where nothing else can be looked upon for relief but radiation. But the radiation must be of the proper nature and must be properly delivered as to place, intensity and amount. This means that we must give the maximum dosage in one treatment, deliver the dosage intratumorally in order to obtain the required intensity to sterilize the mitotic cell, and deliver only the small wave length deep penetrating gamma ray exclusive of all caustic radiation. This we are able to accomplish by the use of the removable platinum radon seed; a seed 4 m.m. in length and filtered by .3 m.m. of platinum—enough filtration to absorb 98.5 per cent of the caustic radiation and to insure the delivery of only gamma radiation—our therapeutic intratumoral radiation. With these seeds we are able to distribute our sources to meet the needs of each case. Each seed delivers 2½ m.c. destroyed or 333 m.c. hours in a period of continuous radiation extending from four to ten days to allow the slow,gradual infiltration of the gamma ray to better accomplish their work of sterilization. Each seed takes care of approximately 4 c.c. of tissue change—and we are able to place as many seeds as each case requires, delivering proper gamma radiation with the assurance that no caustic effect will hinder the therapeutic action of our radiation delivered by the removable platinum radon seed.

Dr. Forbes: I would like very much to add my word of encouragement to what Dr. Harris has said. Dr. Harris has covered the ground very thoroughly, and I am in hearty accord with him. One point, however, I wished to emphasize; that is the lack of a sense reaction which I have observed in the cases where Dr. Herriman has used radium.

The toxic reaction which was the great bugbear during my work with the late Dr. Willis (and Dr. Robinson), who represented the Radium Department of the New York Post-Graduate Hospital, where the twenty-four cases which I reported were treated. If this method of treatment eliminates this distressing element I certainly feel that the patient is entitled to the use of radium in inoperable cases as a palliative measure, and that Dr. Herriman should be encouraged to continue his work and to note his results and embody them in a future-continued report. In my opinion, no harm has resulted to date and many have been made more comfortable.

Injection of Iodized Oil Into the Sphenoid Sinus. Dr. A. Lobell.

(To appear in a subsequent issue of The Laryngoscope.*)*

DISCUSSION.

Dr. T. J. Harris: I wish to congratulate the author of the paper on his very fine work. I have followed what he has been doing with a great deal of interest. The impression he has given us is that so far as the clinical value of the work is concerned it is beyond question. In regard to the approach of the sphenoid, the very minuteness of his technique is of itself a sufficient indication that it is a dangerous procedure. I am not prepared to speak of priority,

but in the last number of Archives of Oto-Laryngology, Dr. Proetz, of St. Louis, presented a far more simple method. His result has been to himself satisfactory; and Dr. Sluder, who has had a larger experience than any other man in America, has used it for the last year. If that is true, if the same results can be secured, or nearly the same results, that method would be preferred to the one advanced tongiht, if for no other reason than because of the delicacy of the procedure.

DR. LEWALD: To supplement what Dr. Harris has said, a method was devised some months ago by Dr. A. W. Proetz and Dr. Edwin C. Ernst, of St. Louis, for introducing fluids into the nasal sinuses without traumatizing their walls. The name, "displacement irrigation," was given to this method. It consists briefly in floating the solution into the nose with the head in such a position that the ostium of the sinus will be inundated. The air is now partially and very slowly exhausted from the sinus by means of a suction apparatus and on releasing the vacuum the fluid enters the sinus. The method was originally devised for treatment but the employment of iodized oils opens a field for accurate X-ray diagnosis. A paper by Dr. Proetz and Dr. Ernst described the positions employed to fill the desired cells and the technic in general was discussed at the meeting of the Radiological Society in Milwaukee, Nov. 29, 1926. Radiographs were used to demonstrate the oil in position in the sphenoid and other sinuses. Whichever method one follows in getting the opaque oil into the sinuses, the information obtained is of very great value, and Dr. Lobell's presentation of a method of studying the sinuses is therefore of interest.

In the early days of pyelography I found difficulty in determining the limits of the normal anatomical pattern of the pelvis of the kidney. We hope that soon Dr. Lobell will be able to tell us of all the variations in the sphenoid sinuses.

DR. LAW: Dr. Lobell has covered this subject in a very complete manner, and has left nothing to add except commendation. While the procedure is not new, having been done at the Manhattan Eye and Ear Hospital before the advent of lipiodol, Dr. Lobell has perfected a technique which makes its use easily available to the rhinologist and with perfect safety, provided there is a previous knowledge of the anatomy as revealed by the X-rays.

It is astonishing sometimes to visualize the shape of a sphenoid and find it nothing like it was supposed to be. I have seen it extend out laterally into the wings of the sphenoid bone in a case where previous X-rays have failed to show its extent. Due principally to the excessively thickened mucosa.

It seems to me the method is superior to the injection by negative pressure, and Dr. Lobell should be encouraged in his efforts to demonstrate the value in selected cases.

There are certain difficulties in the Roentgenographic technique which care, patience and perfect co-operation between the rhinologist and roentgenologist will eliminate.

SECTION ON OTOLOGY.

Regular Meeting, Jan. 14, 1927.

The Newer Tests for Hearing, with Demonstration of Methods. Dr. Edmund P. Fowler.

(Published in full in the April issue.)

The Philosophy of the Older Tests of Hearing. Dr. Robert Sonnenschein.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. WENDELL C. PHILLIPS: I have been promising myself the pleasure of visiting this Section during my presidency of the American Medical Association. You know that for years the work of this Section and that of the Section on Laryngology was combined, and we had only the one Section. I was one of the first agitators and secured enough signatures to induce the Academy to form a Section on Otology, which has well proven the wisdom of

the men to have the Section established. I cannot tell you how much real pleasure I have had during the past year in going about the country and observing what is being done in other cities and towns, not only along the lines which we are following but along all medical lines. I want to say to you men that in the hurry and rush of practice in this great city, I am not altogether sure we are keeping pace with the work that is being done by men in other and smaller towns.

I am promising myself that after the end of this season, which has been a terrific one for me—for I am trying to keep up an active practice and at the same time carry on the duties of the American Medical Association—to come into more active work in this Section.

DR. T. J. HARRIS: The hour is so late that any extensive discussion is quite out of the question, but in the time at my disposal I wish to say just a few things, and especially to add a single word to what Dr. Phillips has said. He is perfectly correct in saying that largely due to his efforts do we owe the founding of this Section, for he sent around the petition that resulted in the founding of this Section. And our Chairman is carrying on this work successfully, for he tells me that at one meeting this winter there were over a hundred present.

Tonight we have had a most unusual meeting. The two speakers have given us a resume of work that was exceedingly illuminating and instructive. Before saying anything further, I wish to speak of what Dr. Sonnenschein and Dr. Fowler have done in this careful study of the registration of sound and the investigation of newer instruments. As I sat here I was thinking how within the last few years we have taken up and carried to an advanced stage the work of the pioneers in otology—Bezold and Politzer and Gruber, and others in the sixties and seventies. Out of this work is coming an inspiration to abandon the slipshod tests of our cases in favor of an accurate method.

One of the points that especially impressed me tonight is the fact that the two speakers have been decidedly together in the fairness with which they have regarded and referred to the older methods—that it is possible to make accurate tests with the tuning fork and that it is possible to make audiograms with the tuning fork. Dr. Fowler tells us that he makes use of the tuning forks as well as the audiometer.

We should not lose sight of the fact that so far as the audiometer is concerned, the work of Dr. Dean, Dr. Bunch, Dr. Jones, Dr. Knudsen and Dr. Fletcher represents something that we should be excessively proud of, for we are far ahead of what is being done on the other side of the water. The demonstration that Dr. Fletcher made tongiht is a speaking witness of the value of it. I was particularly impressed with one feature of great value, the method of testing the hearing of children in the schools. I believe, as Dr. Fowler has said to me, that there are untold possibilities in the work that is going to be done throughout the country in testing the hearing of school children; it is very possible that the early testing of the hearing of children will make a tremendous change in the control of deafness.

Another point I wish to speak of is the availability of the audiometer. It does seem that there should be some way of making it available in the office of every aurist; there should be no prohibition on the score of expense, for it has been well shown tonight that we have in it the means of registering and comparing the hearing from day to day and from week to week, and from office to office that cannot be easily duplicated in any other way. We are all aware of the expense of audiometer today; but I believe the time is not far distant when some way will be devised so that we all will be able to make use of the instrument not only for the study of disease but also for the registering and control of deafness generally.

DR. HARVEY FLETCHER: I don't know what further I can say after this discussion, but there are a few things which I thought I might be expected to comment on tonight, inasmuch as they touch on things in the realm of physics and some helpful suggestions might be in order. Dr. Fowler stated that the results of the old as well as the new methods of testing hearing can be reduced to a single scale. In a paper which I gave before the American Otological Society and also the Academy, I tried to emphasize this fact; for it seems to me that if there is one thing needed, in hearing tests more than any

other, it is that one man's tests can be described to another man in such terms that he can understand. It was for that reason that the unit called "sen" or "su" as used by Dr. Fowler was devised. I wish to emphasize the fact that all the tests which have to do with acuity of hearing can be reduced to a single scale and be expressed in these sensation units. Whether a watch tick, an acoumeter, the voice, a tuning fork, or some source of sound was used in the test, it was pointed out in this paper just how to make the proper reduction. This reduction is sometimes difficult, and when it is difficult it only means that the results from such tests cannot be considered as reliable as those from tests which can be easily reduced to sensation units.

It might be well to point out one or two things which I have observed while repeating these old methods of testing hearing. We carried them out in the laboratory under very quiet room conditions. Let us take for example the common watch tick test. It would be absurd to say that it has no practical value. It has, as most of you can testify. However, it cannot be considered as an accurate test of the hearing. In a place where there are no sound reflections, the intensity of the sound varies inversely as the square of the distance from the watch. (Diagram on board and demonstration.) We never can find such an ideal place. Even though there are no reflections from the walls of the room, reflections from the head of the patient occur. Measurements in a soundproof room which was heavily damped with curtains showed that the watch was louder at 12 inches than at 6 inches. That is not an unusual thing for a watch tick, or a coin click, or acoumeter, especially in an ordinary room. In my opinion, as much error has been made in assuming that the distance variations are directly related to intensity variations as in assuming a constant intensity in the sound source used.

An untold amount has been written about how to strike a tuning fork so as to give it a constant intensity; but little was said about the damping properties of the work. It seems to me that they were straining at a gnot and swallowing a camel. So Dr. Fowler's comments along this line are very timely. In starting a tuning fork you need to worry more about how you hold it, for variations from this source are more than those arising from striking the fork. We have found by actual measurement that you get a large variation in intensity from a fork from just a little turning it around at an angle or variations in position, much more than you get from variations in striking it.

You might be interested to know the deafening effect of putting your finger in the ear. It is surprisingly small. Putting the finger in the ear will reduce the hearing ability only 20 sensation units. Of course when you talk in terms of intensity, the change sounds much greater, being a reduction to 1 per cent of its uninterrupted value. If the difference in the hearing loss of the two ears is only 20 units, stopping the good ear with the finger does not insure that the sounds will be heard in the bad ear.

Regarding the question of the intensity from the prongs of the fork or the handle of the fork — which was mentioned by Dr. Sonnenschein; of course the intensity of the handle or the prongs of the fork depends entirely on your point of view. For communicating sound energy from the fork to the air, either one is very inefficient. The tuning fork is about the worst thing you could use for communicating sound to the air. Why? Because you have a heavy, rigid body trying to stir up a very light, flexible body. It is like trying to communicate a lot of energy from your moving fist to a light feather. If you want to communicate sound energy to a solid material like a desk, you put the handle of the fork on it; and under such circumstances, an efficient transfer takes place. So it seems to me to talk about one or the other having the greater intensity, the purpose for their use must be kept in mind.

UNIVERSITE DE PARIS.

Faculté De Médecine.

COURSES ON BRONCHOESOPHAGOSCOPY AND ANAPLASTIC SURGERY OF THE FACE AND NECK.

1. *Course on Bronchoesophagoscopy* will be given by Professor Chevalier Jackson, of Philadelphia, on the joint invitation of Professor Pierre Sébileau and the Faculté de Médecine.

This course will consist of five sessions: *First session:* (Monday, July 11, 1927). Technique of Direct Laryngoscopy, Bronchoscopy, Esophagoscopy and Gastroscopy.

Second session: (Tuesday, July 12, 1927). Foreign bodies in the air and food passages; pathology, symptomatology and clinical diagnosis; Roentgen-ray diagnosis of nonopaque foreign bodies.

Third session: (Wednesday, July 13, 1927). Mechanical problems encountered in the peroral bronchoscopic, esophagoscopic and gastroscopic removal of all kinds of foreign bodies from the air and food passages.

Fourth session: (Friday, July 15, 1927). Diseases of the bronchi and lungs. Bronchoscopic aid to the internist and surgeon in the diagnosis and treatment of suppurative and malignant diseases of the lungs. Post-tonsillectomic pulmonary abscess. Peroral bronchoscopic aspiration of pus from the lungs in bronchiectasis, pulmonary abscess, and drowned lung. Technique of removal of specimens of tissue for biopsy and of secretions for vaccine therapy.

Fifth session: (Saturday, July 16, 1927). Application of endoscopy to the diagnosis and treatment of diseases of the esophagus.

These five sessions of the course in Bronchoesophagoscopy will be held in the morning at 10 o'clock, at the Oto-Rhino-Laryngologic Clinic, Hopital Lariboisiere, Service of Professor Pierre Sébileau. Each session will require about two hours. The course will be given in English, with occasional interpretations in French by M. le Dr. Edmond Aucoin. "Chalk talk," lantern slides, the open manikin board and the cinematograph will be used in the demonstrations.

The course of five sessions by Professor Chevalier Jackson will be accompanied and followed by a practical course in Bronchoesophagoscopy to be given from July 11 to July 30, in French and English, by Doctors Edmond Aucoin and Fernand Eeman, assistants of Professor Chevalier Jackson.

First week (July 11 to July 15, 1927). Cadaver work on endoscopic anatomy and landmarks; the position of the patient; the technique of direct laryngoscopy, bronchoscopy and esophagoscopy; the dangers and how to avoid them; endoscopic operations. The mechanical problems presented by various classes of foreign bodies are demonstrated and the registrants are trained in the best and safest methods of removal.

Second week (July 18 to 23, 1927). Bronchoscopic and esophagoscopic work in the living, moving bronchi and esophagus and stomach of the narcotized dog.

Third week (July 25 to 30, 1927). Clinical demonstrations and endoscopic procedures.

The five theoretic sessions and the clinical seances take place at 10 o'clock in the morning at the Oto-Rhino-Laryngologic Clinic of the Faculté de Médecine (Service of Professor Pierre Sébileau), at the Hopital Lariboisiere.

The practical sessions of the Bronchoesophagoscopic course take place each day at 2 p. m. (14 o'clock) at the Laboratoire de Médecine Operatoire of the Faculté de Médecine (Professor B. Cunéo) and require about two hours.

The five sessions of Professor Chevalier Jackson are gratuitous and are open to all practitioners and students. Registration is unnecessary.

The practical course is limited to six registrants; the fee for the laboratory of the Faculté de Médecine is 500 francs. To register for this course address: M. le Doyen, Faculté de Médecine, Paris.

2. *Course in Anaplastic Surgery of the Face and Neck,* to be given by Professor Pierre Sébileau and Dr. Leon Dufourmentel, Chief of Clinic.

This course of reparative and cosmetic surgery of the face and neck consists of 12 theoretic and 12 practical sessions.

First session (Friday, Sept. 16, 1927) by Dr. L. Dufourmentel. Cosmetic surgery of the skin. (Dissimulated incisions; suturing; dressings; treatment of cicatrices; surgical tattooing.)

Second session (Sautrday, Sept. 17, 1927) by Professor Pierre Sébileau. Reparative surgery of the skin. (Skin-grafts and skin-flaps.)

Third session (Monday, Sept. 19, 1927) by Dr. L. Dufourmentel. The surgical leveling of the skin by use on the skin and soft parts. Leveling after distension (wrinkled skin; atony), flaccidity of facial paralysis; depressions following accidental or surgical trauma; ptosis, cutaneous pouches. Grafts and filling flaps of muscle, fat, aponeurosis.

Fourth session (Tuesday, Sept. 20, 1927) by Prof. Sébileau. Surgical leveling of the skin by organic support. Grafts, osteoperiosteal and cartilaginous flaps. Osteoplasty, osteotransplantation. Grafts of continuity; treatment of pseudoarthroses of the lower jaw. Filling grafts for osseous cavities and treatment of loss of substance of the cranium and face.

Fifth session (Wednesday, Sept. 21, 1927) by Dr. L. Dufourmentel. Surgical correction of nasal deformities. Treatment of

skeletal depressions, deviations, torsions, hypertrophies, projections, elongations and enlargements of the nasal pyramid.

Sixth session (Thursday, Sept. 22, 1927) by Dr. L. Dufourmentel. Surgical correction of partial nasal destruction; partial rhinoplasty. Repair of the lobule, subseptum and alae.

Seventh session (Friday, Sept. 23, 1927) by Dr. L. Dufourmentel. Surgical correction of total and subtotal destructions of the nasal pyramid (regional rhinoplasty). Method of placing osseous and cartilaginous grafts "en nourrice." Repair of the nasal skeleton. Method of superposed flaps.

Eighth session (Saturday, Sept. 24, 1927) by Dr. L. Dufourmentel. Restoration and correction of the lips (cheilorrhaphy and cheiloplasty. Restoration of mucosal covering. Restoration of the planes of thickness and of cutaneous covering. Commisural enlargement. Correction of deformities, hypertrophies, irregularities, cicatricies).

Ninth session (Monday, Sept. 26, 1927) by Professor Sébileau and Dr. L. Dufourmentel. Opercular restoration of large losses of substance of the buccal walls. Reparative surgery of the chin. Practice of bipedicular flaps of the neck and scalp. Application of these flaps on inorganic supports. Turned and superposed flaps.

Tenth session (Tuesday, Sept. 27, 1927) by Professor Pierre Sébileau . Surgical treatment of congenital clefts of the hard palate and velum. (Urano-staphylorraphy).

Eleventh session (Wednesday, Sept. 28, 1927) by Dr. L. Dufourmentel. Surgery of the temporomaxillary articulation. Surgical treatment of irreducible and recurrent luxations and of ankyloses of the temporomaxillary articulation. Correction of prognathism and of mandibular deviations.

Twelfth session (Thursday, Sept. 29, 1927) by Prof. Pierre Sébileau and Dr. L. Dufourmentel. Correction of deformities of the external ear. Treatment of salivary fistulae. Technique of cosmetic operations on the auricle. Drainage and resection of salivary fistulae. Enervation of the parotid gland.

The clinical and theoretic parts of the course in anaplastic surgery will take place at 10 a. m. in the Oto-Rhino-Laryngologic Clinic of the Hopital Lariboisiere.

The technical part will take place at 2 p. m. (14 o'clock) at the Amphitheatre d'Anatomie, 17 Rue du Fer a Moulin.

To register for the gratuitous theoretic and clinical course, address: M. le Doyen, Faculté de Médecine, Paris. (No fee required.)

For the practical course in anaplastic surgery the registration fee is 1000 francs. Address: Secretariat, Amphitheatre d'Anatomie, 17 Rue du Fer a Moulin, Paris.

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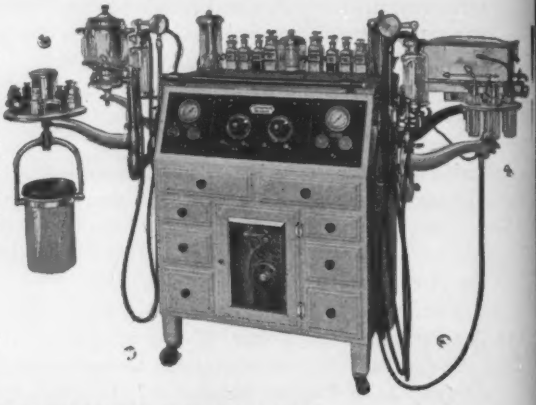

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